COMPARING THE ACCURACY OF 12HOURS URINE PROTEIN WITH 24 HOURS URINE PROTEIN IN PREDICTING PROTEINURIA IN HYPERTENSIVE DISORDERS OF PREGNANCY

A Dissertation Submitted to

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

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In Partial Fulfilments of the Regulations

for the Award of the Degree of

M.S. (OBSTETRICS & GYNAECOLOGY) - BRANCH – II

GOVERNMENT STANLEY MEDICAL COLLEGE

CHENNAI

April - 2014

BONAFIDE CERTIFICATE

This is to certify that this dissertation is the bonafide work of Dr.P.NIRMALADEVI on **"COMPARING THE ACCURACY OF 12HOURS URINE PROTEIN WITH 24 HOURS URINE PROTEIN IN PREDICTING PROTEINURIA IN HYPERTENSIVE DISORDERS OF PREGNANCY"** during her M.S.,(Obstetrics and Gynaecology) course from April 2011 to April 2014 at the Government Stanley medical college and Raja Sir Ramasamy Mudaliar Lying-in Hospital,Chennai.

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DECLARATION

I, **Dr.P.Nirmala Devi**, solemnly declare that the dissertationtitled **"COMPARING THE ACCURACY OF 12HOURS URINE PROTEIN WITH 24 HOURS URINE PROTEIN IN PREDICTING PROTEINURIA IN HYPERTENSIVE DISORDERS OF PREGNANCY"** is done by me at RSRM Lying in Hospital ,Stanley Medical College and Hospital under the guidance of PROF DR.V.KALAIVANI M.D;D.G.O; Professor and Head of the Department of Obstetrics and Gynaecology, Stanley Medical College& RSRM Lying in Hospital, Chennai 13.

This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of MD degree Branch II (Obstetrics and Gynaecology).

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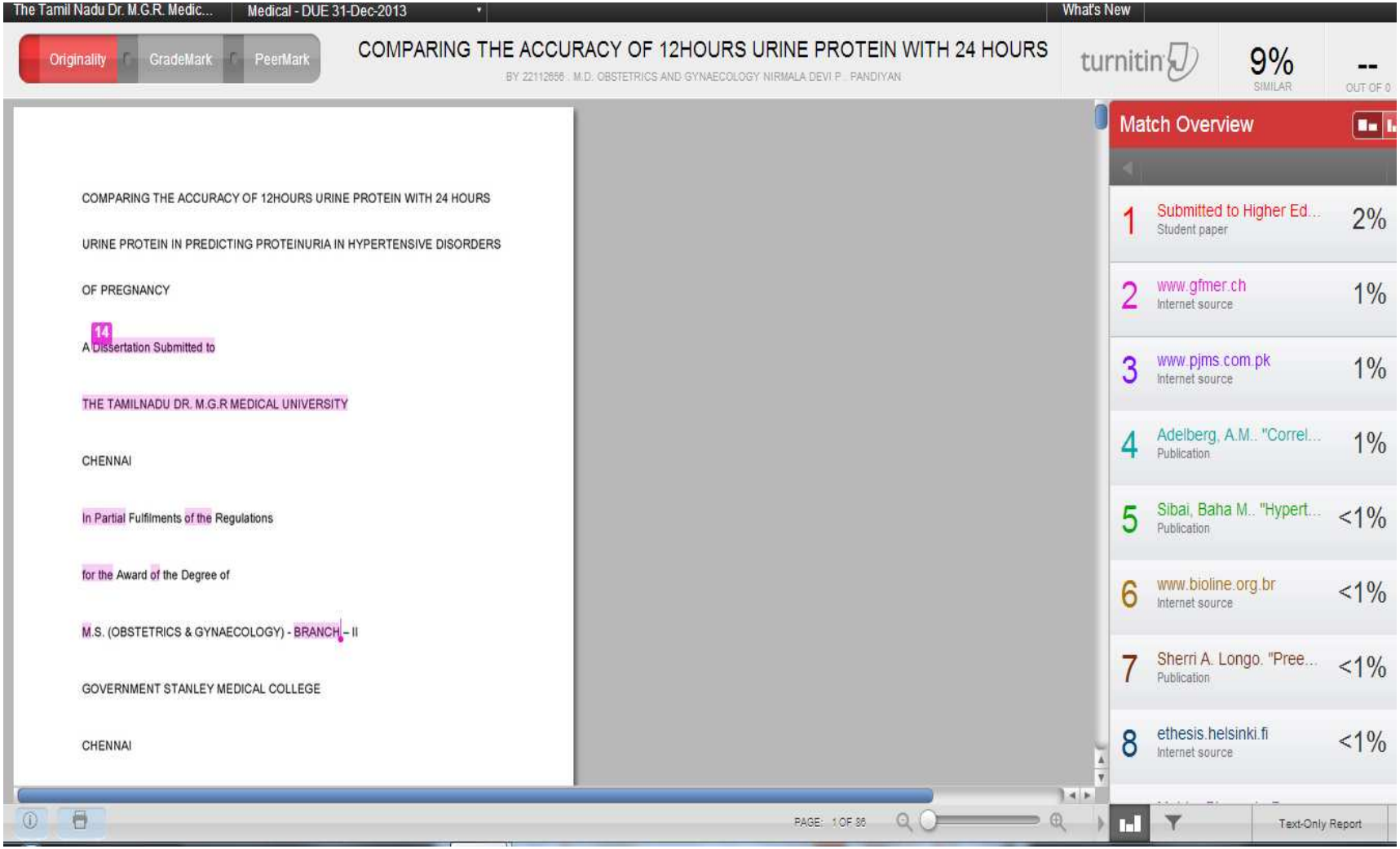
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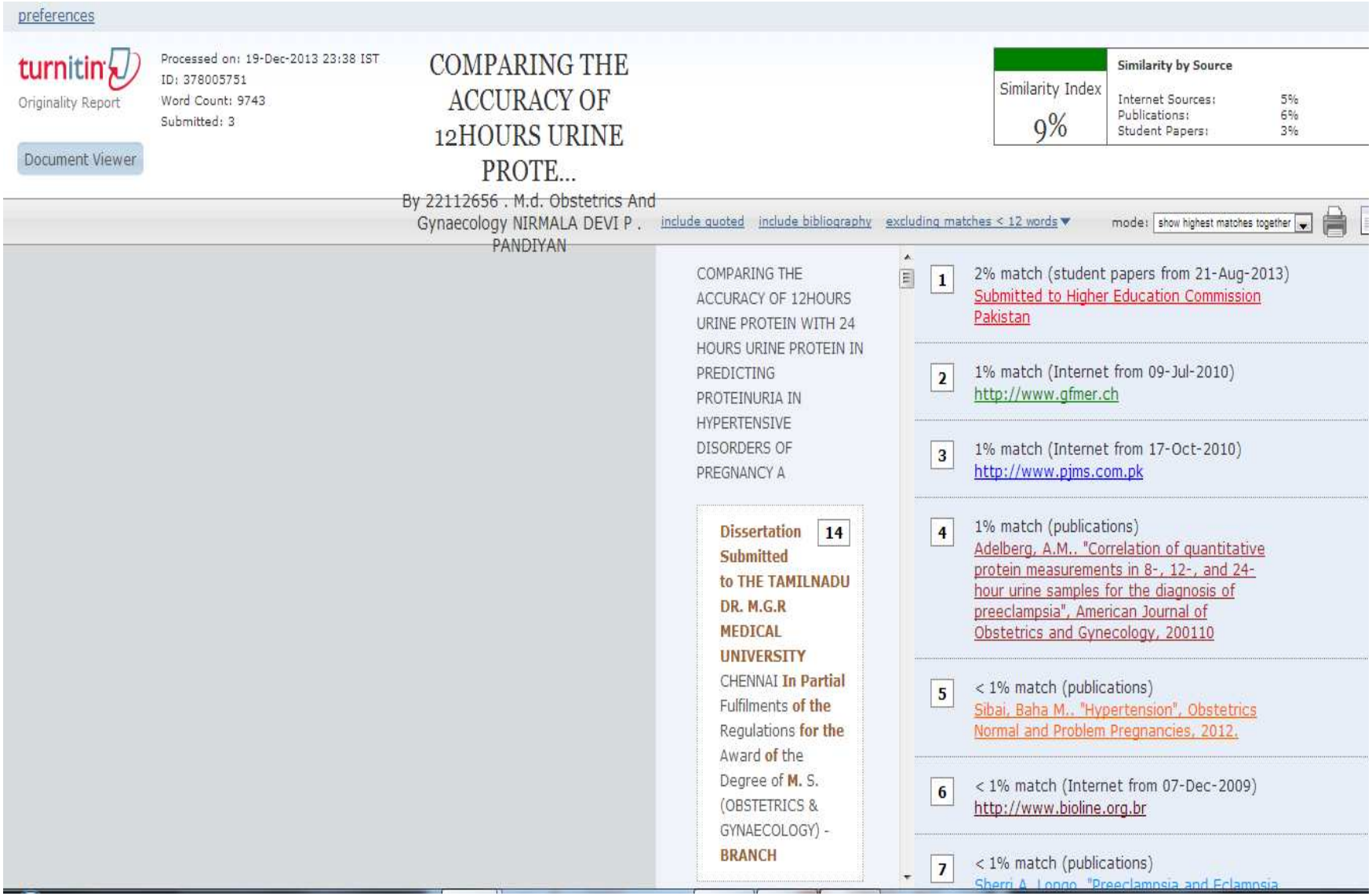
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I.INTRODUCTION

Pregnancy brings happiness to mothers but at times brings hypertension to mothers**.**Hypertensive disorders complicating pregnancies forms one of the deadly triads along with hemorrhage and infections that results in much of the maternal morbidity and mortality in pregnancy.Hypertensive disorders complicate 5-10% of all pregnancies. WHO systematically reviews maternal mortality worldwide and found that 16% of maternal deaths are due to hypertensive disorders.The mystery how pregnancy incites or aggravates hypertension is still unresolved.

Pre-eclampsia is diagnosed when hypertension and proteinuria occur after 20 weeks of gestation. Edema is often seen but is not required to make the diagnosis. Proteinuria is an important sign of preeclampsia. It reflects the glomerular damage that causes leakage of proteins through the basement membrane.

There are so many components which determine the severity of hypertension. Of which proteinuria is important to classify the type of hypertension. Presence of proteinuria differentiates gestational hypertension and preeclampsia.It is directly proportional to maternal and perinatal morbidity and mortality. Severity of preeclampsia is also determined by the degree of proteinuria hence accurate and rapid quantification of proteinuria is essential for the diagnosis and management of hypertensive disorders.

Routinely proteinuria in pregnancy is detected by urine dipstick

Method which is not accurate and there are false positives and false negatives. The gold standard 24hrs urine protein though accurate is time consuming and requires patient compliance which can be rectified by shorter collection specimens (12hrs) which gives similar results as 24 hrs urine protein.

II. REVIEW OF LITERATURE

Pregnancy can cause hypertension in previously normotensive women and can aggravate already existing hypertension. Preeclampsia is a pregnancy specific disorder which leads toplacental dysfunction and maternal complications. Preeclampsia canmanifest either as a maternal syndrome or as a fetal syndrome. Thehallmark of preeclampsia is hypertension along with proteinuria[1]. Edema is not an essential criteria for the diagnosis of preeclampsia.

Hypertension in pregnancy can be defined as

- Systolic Blood Pressure $\geq$ 140 mm Hg
- Diastolic Blood Pressure $\geq$ 90 mm Hg

Recorded on two occasions six hours apart within seven days [2].Blood Pressure should be recorded in sitting posture with right arm well supported in horizontal position at the level of heart.

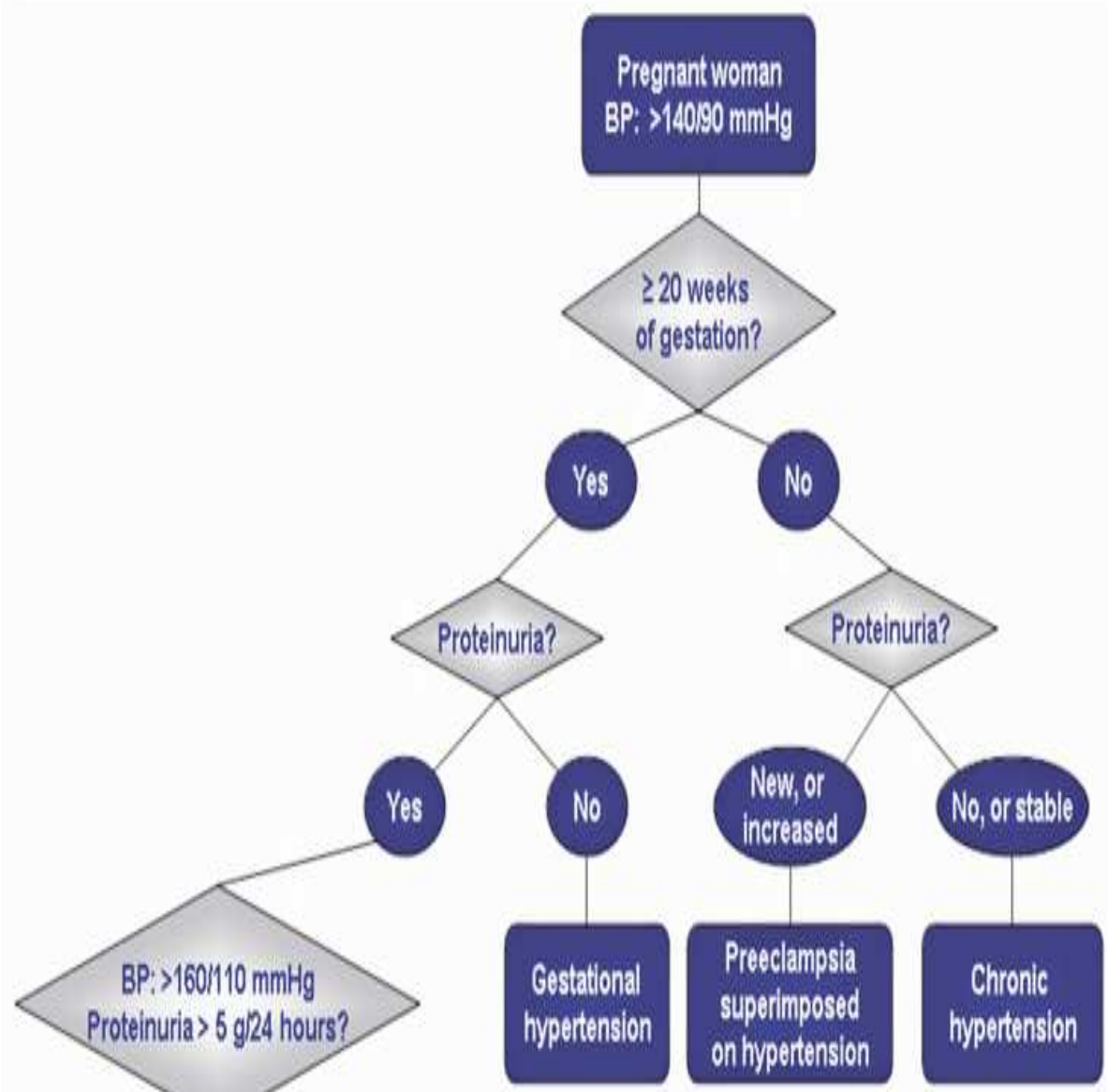
GOLD STANDARD for the measurement of blood pressure is by mercury sphygmomanometer. An adequate size cuff isUsed. Korotkoff V is used as an index of Diastolic blood pressure.

"PROTEINURIA in hypertensive disorders of pregnancy is definedas >300mg of protein excretion in a 24 Hrs urine sample. Spot Protein Creatinine

Ratio > 0.3 mg. Dipstick reading > 1+ in two random urine samples collectedsix hours apart." [3]

HISTORICAL ASPECTS OF PREECLAMPSIA:

- Defined as toxaemia by ZWEIFF.
- Defined as Disease of Theories by CHESLEY AND ZWEIFF.
- Relation of Essential Hypertension to preeclampsia described by FISHBERG.
- Discovery of proteinuria in preeclampsia by LEVER AND SIMPSON.
- Physiological and functional changes in glomeruli described by LABLEIN AND FAHR.
- Electron Microscopic demonstration of Glomerular Capillary Endotheliosis by FARGUHER.



Diagnostic Criteria for Preeclampsia and Severe Preeclampsia

	Systolic BP (mmHg) [a]	Diastolic BP (mmHg) [a]	Proteinuria (g / 24 hrs)	
Preeclampsia	> 140	>90	> 0.3	[a]Two separate BPs at least 4 hours apart
Severe preeclampsia	> 160	> 110	> 5.0	

CLASSIFICATION:

There are various classifications for hypertensive disorders of pregnancy. Due to lack of knowledge of the precise nature of thedisorder and difference of opinion in the clinical and pathologicalfeatures an agreed classification is yet to put forward.

The Worldwide accepted classification is theNATIONAL HIGH BLOOD PRESSURE EDUCATION PROGRAMMEoftheworking group (2000) [4]. According to that Hypertensive disorders areclassified as

- Gestational Hypertension
- Preeclampsia
- Eclampsia
- Preeclampsia superimposed on chronic Hypertension
- Chronic Hypertension.

GESTATIONAL HYPERTENSION:

Also called as Transient Hypertension of pregnancy. “Blood Pressure ≥140/90 mm Hg recorded for the first time during pregnancy after 20 weeks of gestation.No associated proteinuria. Blood pressure becomes normal within 12 weeks postpartum[5].Retrospective diagnosis made postpartum.”

PREECLAMPSIA:

The hallmark of preeclampsia is Hypertension associated with proteinuria developing after 20 weeks in a previously normotensive woman.

MINIMUM CRITERIA: "Blood pressure ≥ 140/90 mmHg recorded for the first time after 20 weeks of gestation.Proteinuria of ≥ 300mg in 24 Hrs or > 1+ dipstick."

ACOG Classifies preeclampsia as [6]

Features	mild/non severe	Severe
diastolic blood pressure	< 110mmhg	≥110 mmhg
systolic blood pressure	< 160mmhg	≥160mmhg
Proteinuria	≤2+	≥3+
headache/ visual disturbances	Absent	Present
epigastric pain/oliguria	Absent	Present
serum Creatinine	Normal	>1.2mg/dl
Thrombocytopenia	Absent	<1,00,000
pulmonary edema	absent	Present
iugr/ oligohydramnios	Absent	Present
ast/ alt	minimally elevated	marked elevation
Ldh	Absent	ldh >600

ECLAMPSIA:

Occurrence of tonic clonic seizures in a women with Preeclampsia which is not attributable to any other cause.

PREECLAMPSIA SUPERIMPOSED ON CHRONIC HYPERTENSION[7]:

"New Onsetof Proteinuria>300mg/ 24 hrs in ahypertensivewomen with no proteinuria before 20weeks,

Sudden increase in proteinuria or blood pressure or fall in platelet count< one lakh in a hypertensive women with proteinuria before 20 weeks."

CHRONIC HYPERTENSION:

"Blood Pressure > 140/90mmHg before pregnancy or diagnosed before 20weeks of gestation.

Hypertension first diagnosed after 20 weeks but persists after 12 weeks postpartum."

INCIDENCE:

- Gestational Hypertension – 5%
- Preeclampsia –Williams :5-7%
- Mudaliar and Menon : 8-10% [8]
- De Sweit : 3-7%

- Primi gravida- 7%
- Multigravida -5%
- Superimposed preeclampsia -25%
- Chronic Hypertension -1-2%
- Eclampsia -0.5-2%

RISK FACTORS FOR PREECLAMPSIA

PRIMIGRAVIDA:

According to McGillivray preeclampsia occurs in primi gravida 15 times more commoner than parous women.

PRIMIPATERNITY:

There is a inverse relationship between the partner and occurrence of preeclampsia. Hence not only the first pregnancy is a risk factor but also the first pregnancy by the existing partner. [9 – 14]Recent epidemiologic studies suggest that multiparous women withdifferent partners have a higher risk for pre-eclampsia than domultiparous women with the same partner, perhaps because of a protective effect of repeated exposure to specific antigens.

INCREASING MATERNAL AGE:

Robillard and Haley et al states that an extreme of age is a risk factor for the development of preeclampsia.

FAMILY HISTORY:

There is a genetic relationship in the development of preeclampsia. Daughters of mothers who had preeclampsia has a 25% risk of developing preeclampsia.

RECURRENT PREECLAMPSIA:

Recurrence risk is between 2 -25% [15]

OBESITY:

SIBAI et al showed that obesity perse is a independent riskfactor for the development of Hypertension. It also forms a component of metabolic syndrome which increases the risk of preeclampsia.

MEDICAL DISORDERS:

- Diabetes increases the risk by 30%.
- Chronic Hypertension increases the risk by 3-7%, [18]
- Antiphospholipid antibody syndrome and thrombophilias had a strong correlation with preeclampsia.[19 – 21]

RISK FACTORS FOR PREECLAMPSIA

COUPLE RELATED RISK FACTORS:

- Prim paternity and limited sperm exposure
- Pregnancy after artificial insemination
- Protective effect of partner change in case of previous preeclamptic pregnancy

MATERNAL OR PREGNANCY RELATED RISK FACTORS:

- Extremes of maternal age
- Multifetal gestation, preeclampsia in previous pregnancy
- Chronic hypertension or renal disease
- Antiphospholipid antibody syndrome, thrombophilias
- Obesity and diabetes
- Family history of preeclampsia

THEORIES REGARDING CAUSATION OFPREECLAMPSIA:

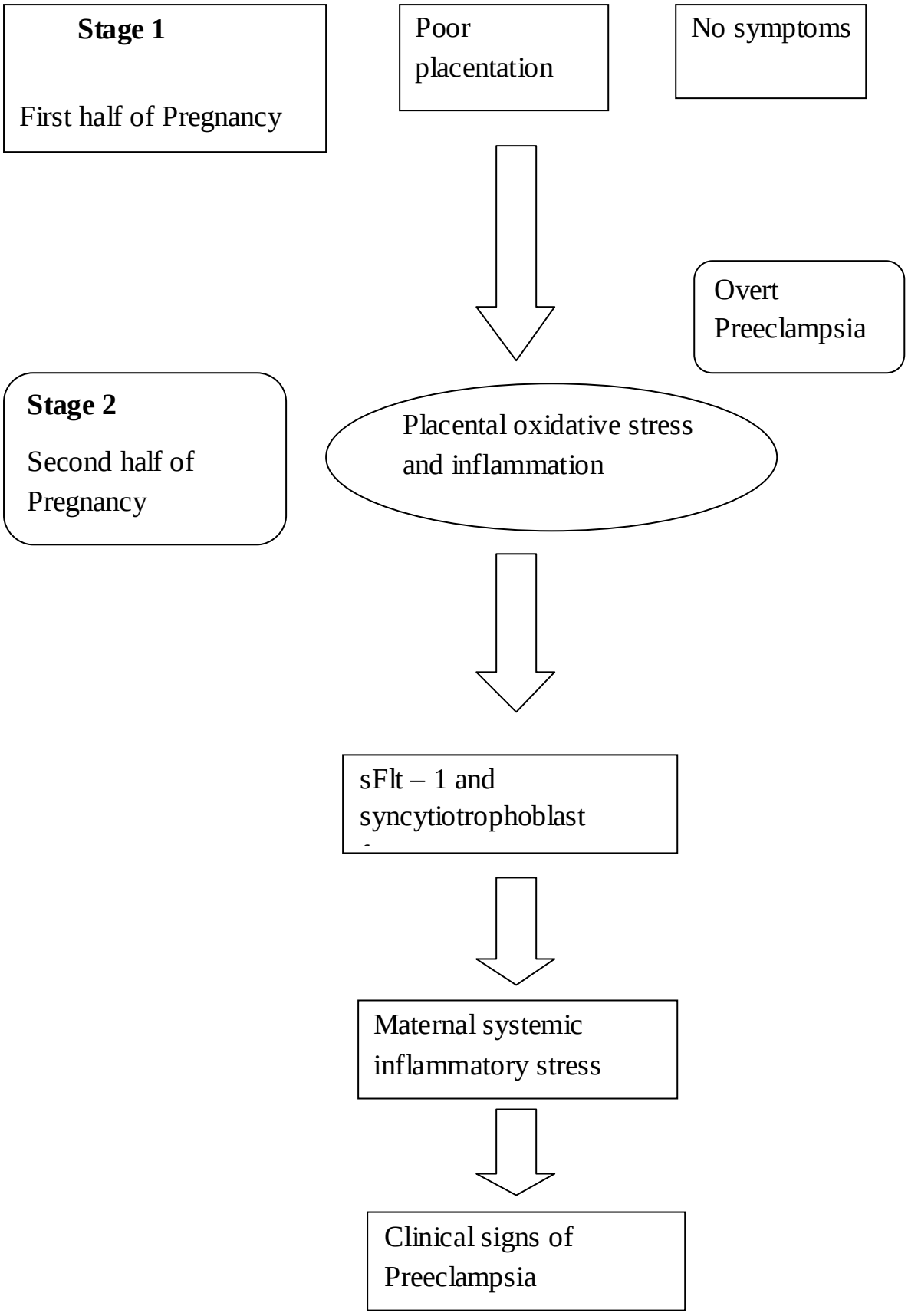
According to various theories regarding the pathophysiology of preeclampsia a women is likely to develop hypertensive disorders during pregnancy when

- She is exposed for the first time to chorionic villi, [25]
- She is exposed to abundance of chorionic villi like molar pregnancy and multiple pregnancies.
- When she already has vascular disease,
- There is genetic predisposition.

TWO STAGE HYPOTHESIS:

According to this hypothesis there occurs poor placentation due to faulty endovascular trophoblastic remodelling which occurs during first half of pregnancy (stage 1) which leads to poor placentation leading to (stage 2) clinical syndrome which is characterised by activation of maternal systemic inflammatory stress and clinical signs of preeclampsia.

THE TWO STAGE MODEL OF PRE-ECLAMPSIA:



ABNORMAL TROPHOBLASTIC INVASION:

During pregnancy there occurs a physiological change in the spiral arterioles which convert them into uteroplacental arteries which occurs in two stages. During the first stage there occurs endovascular trophoblastic invasion of the decidual segment of the spiral arterioles. During 16 to 20 weeks of gestation there occurs secondary trophoblastic invasion of the myometrial segment of spiral arterioles which converts them into more elastic tortuous easily distensible channels[26 -27].

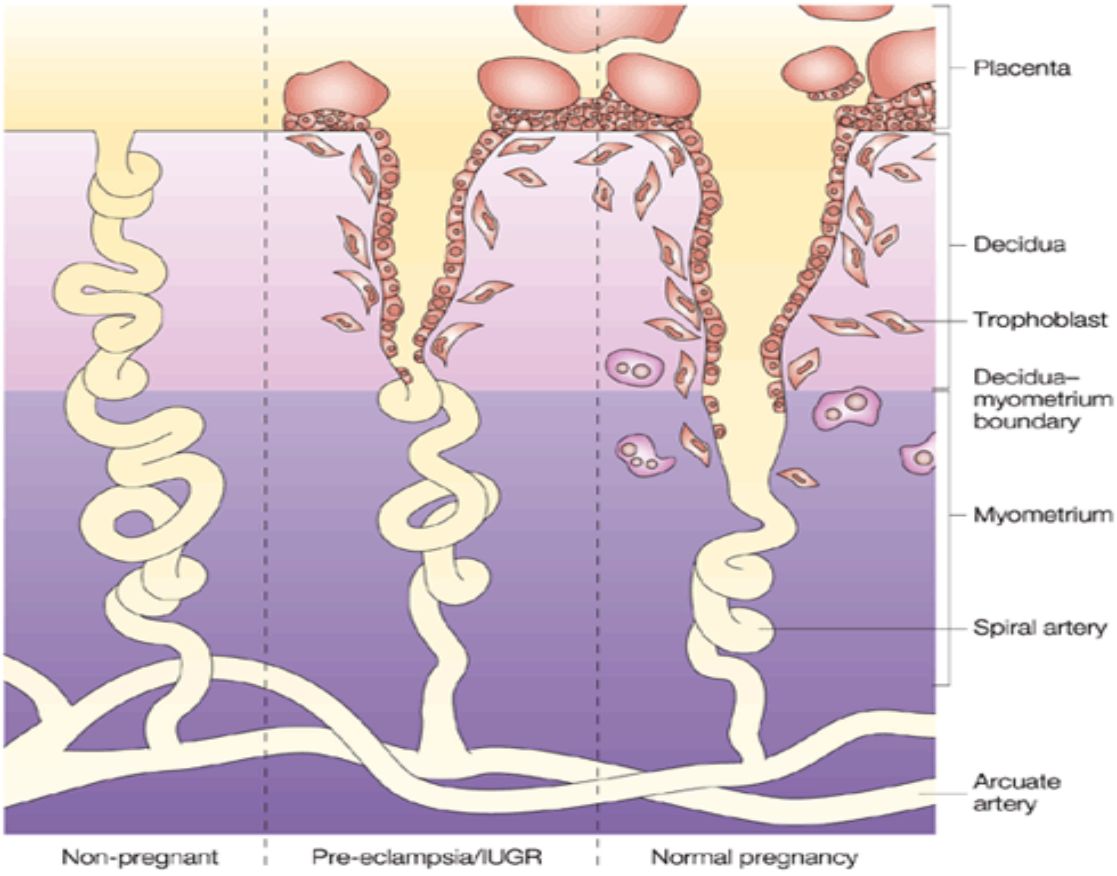


FIGURE:1: Trophoblastic Invasion

In preeclampsia these vascular changes does not occur and the myometrial segments of the spiral arterioles are left with muscular lining which leads to decreased distensability and becomes hyper responsive to the hormonal changes.[28-29]

IMMUNOLOGICAL THEORY:

There is a disturbance in the immunological balance between the maternal and fetal system in preeclampsia which interferes with the remodelling of spiral arterioles by the trophoblastic cells which leads to fetoplacental hypoperfusion.[30]

In preeclampsia there occurs imbalance between the formation of maternal antibodies and the fetal antigenic load and the formation of blocking antibodies is impaired (BARDEQUE and associates 1999).In preeclampsia the immune system is hyper responsive since the normal immunosupression which prevents the fetal allograft rejection is impaired (LABARRERE 1998)[31].

T Lymphocytes levels are lower in preeclampsia [32] which leads to decreased levels of cytokines which leads to poor implantation and dysfunction (HYASHI and associates 2000).[33-34]

HLA-G which is a immunosuppressant is expressed by cytotrophoblast and protect the trophoblast from destruction by the NK Cells.In a women with preeclampsia the

level of HLA-G is low and recognition of Fetal cytotrophoblast as foreign antigens[35 -36].

GENETIC PREDISPOSITION:

NISSAN et al showed that there is a strong genetic predisposition of

31% in Preeclampsia[37].

- Hypertensive disorders of pregnancy are inherited both by single gene and multifactorial inheritance (BROUGHTON PIPLUS 1999)[40].
- Single recessive model with a frequency of 0.25% with Genes acting on mother on ch 1, 3, 9.

Dominant model with incomplete penetrance.

- Mother to daughter transmission is through HLA DR4.(KILPATRIK et al) [41].
- Higher incidence in women carrying Angiotensinogen gene variant T235 (ARNGRIMSSON et al)[42] .
- Association between proteinuric hypertension and HLADR4 has been reported.
- Incidence of factor V Leiden mutation polymorphism variant 20210A.

- Epigenetic features or imprinting locus on ch 10q22 is also involved (DUDEJANS et al).Increased level of MTHFR gene mutation homozygosity (COFFER et al) [44]

ENDOTHELIAL DYSFUNCTION:

The endothelium plays a major role in the pathophysiology of preeclampsia indicated by the imbalance between prostacyclin and thromboxane ([40 – 42]) and decreased synthesis of nitric acid. In preeclampsia endothelial cell dysfunction and platelet aggregation precedes increase in thrombi and fibrin formation [43 – 46].

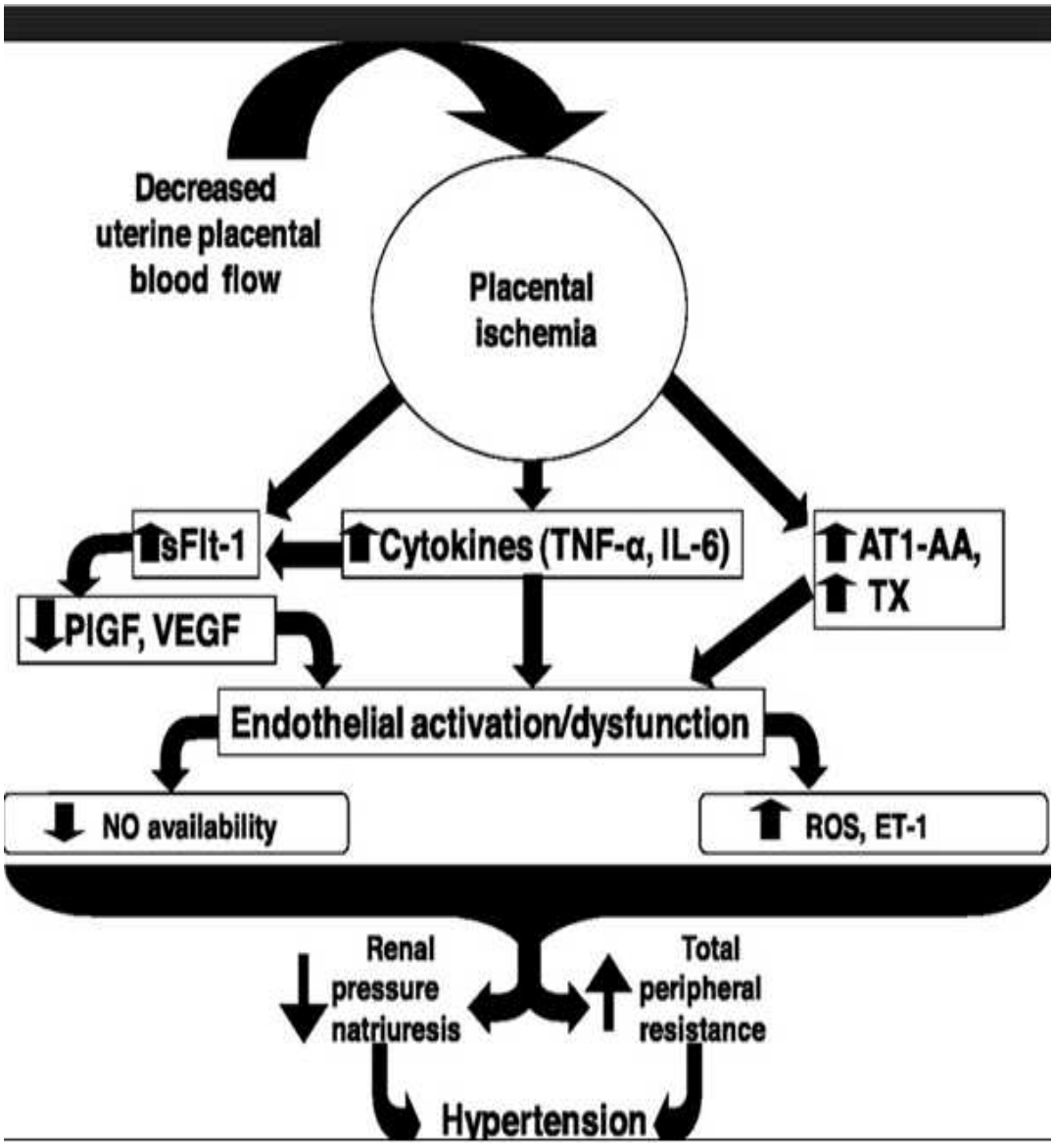
According to MAYNARD et al [47]) in preeclampsia there is increased production of soluble fms like Tyrosine kinase 1(sflt-1) by the placenta .It binds to the receptor binding domain of Vascular Endothelial Growth Factor(VEGF) and Placental Like Growth Factor (PLGF) leading to decreased levels of VEGF and PLGF resulting in endothelial dysfunction [48 -50].

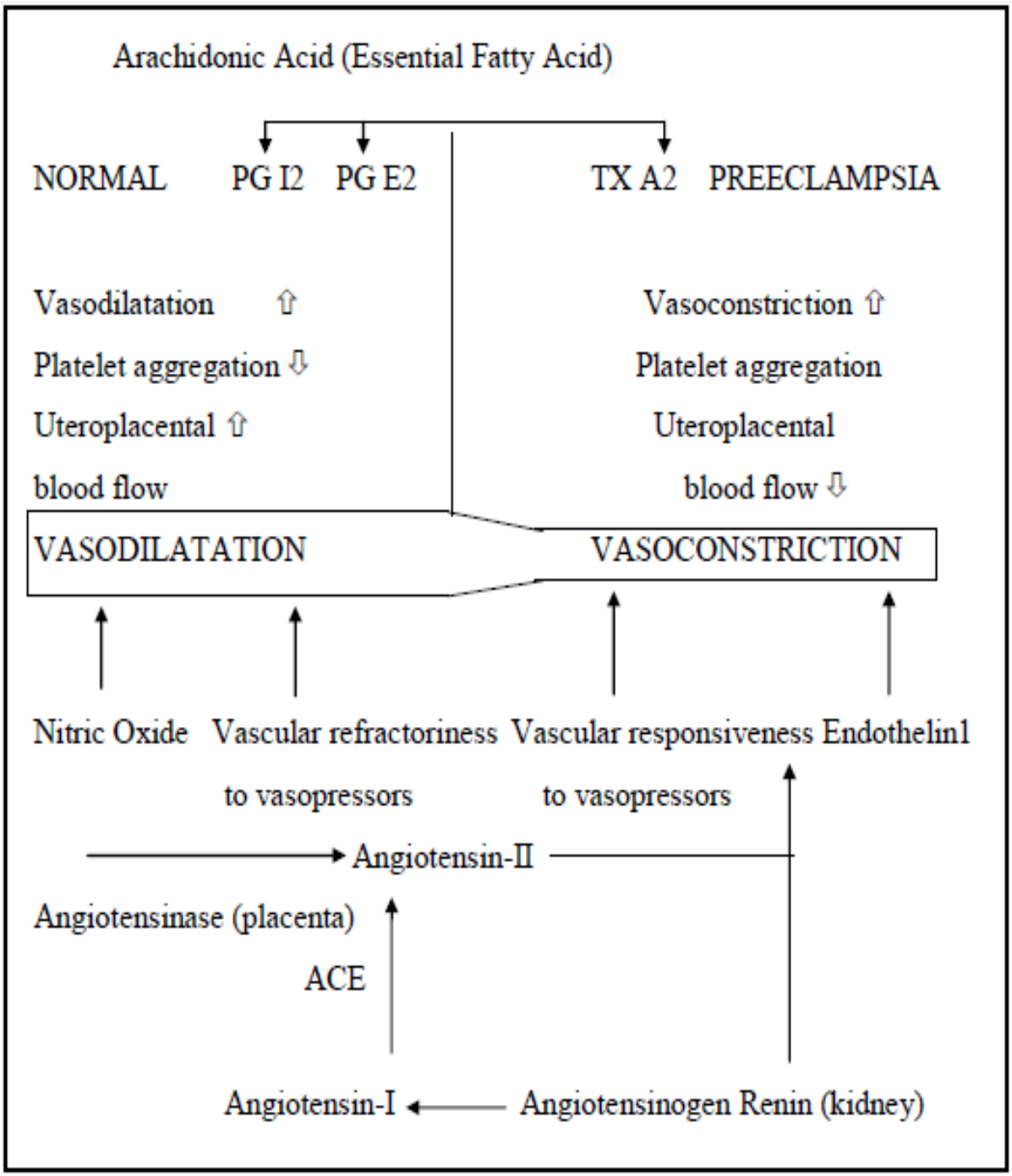
According to LEVINE et al [57 -58] there is increased levels of soluble Endoglins S (Eng) in preeclampsia which decreases Nitric oxide production leading to vasoconstriction and preeclampsia.

Deficient trophoblastic invasion which is immunologically mediated leads to release of placental factors into maternal circulation and causes damage to the vascular endothelium [60].

WANG et al [61] studied the imbalance between TXA2 and PGI2 with increased levels of TXA2 leading to vasoaggregation and endothelial damage.

Endothelin -1 a potent vasoconstrictor level is increased in preeclampsia (CLARK SCHIFF) [62].




Arachidonic Acid (Essential Fatty Acid)
NORMAL
PG I2
PG E2
TX A2
PREECLAMPSIA
Vasodilatation ⇧
Platelet aggregation ⇩
Uteroplacental ⇧
blood flow
Vasoconstriction ⇧
Platelet aggregation
Uteroplacental
blood flow ⇩
VASODILATATION
VASOCONSTRICTION
Nitric Oxide
Vascular refractoriness to vasopressors
Vascular responsiveness to vasopressors
Endothelin1
Angiotensin-II
Angiotensinase (placenta)
ACE
Angiotensin-I
Angiotensinogen
Renin (kidney)

ENDOTHELIAL FACTOR:

Endothelial cell activation is the central event in the pathophysiology

of preeclampsia. The normal endothelium is lined by a single layer Of squamous epithelial cells which lines the vascular lumen which functions as follows

- It plays an important role in haemostasis and decreases vascular smooth muscle response to vasopressors.
- It has anticoagulant properties and prevents clot formation.
- When the endothelium is damaged there occurs increased coagulation due to increased sensitivity to pressor agents.

NITRIC OXIDE:

Endothelium in normal pregnancy produces Nitric Oxide which is a derivative of L-Arginine is a potent vasodilator which maintains a low pressure low resistance state in the fetoplacental unit [63].

There are various studies which shows decreased Nitric Oxide levels in preeclampsia. WANGand colleagues' 2004 showed an increased level of Nitric Oxide in severe preeclampsia which is a compensatory process which signifies worsening of disease.

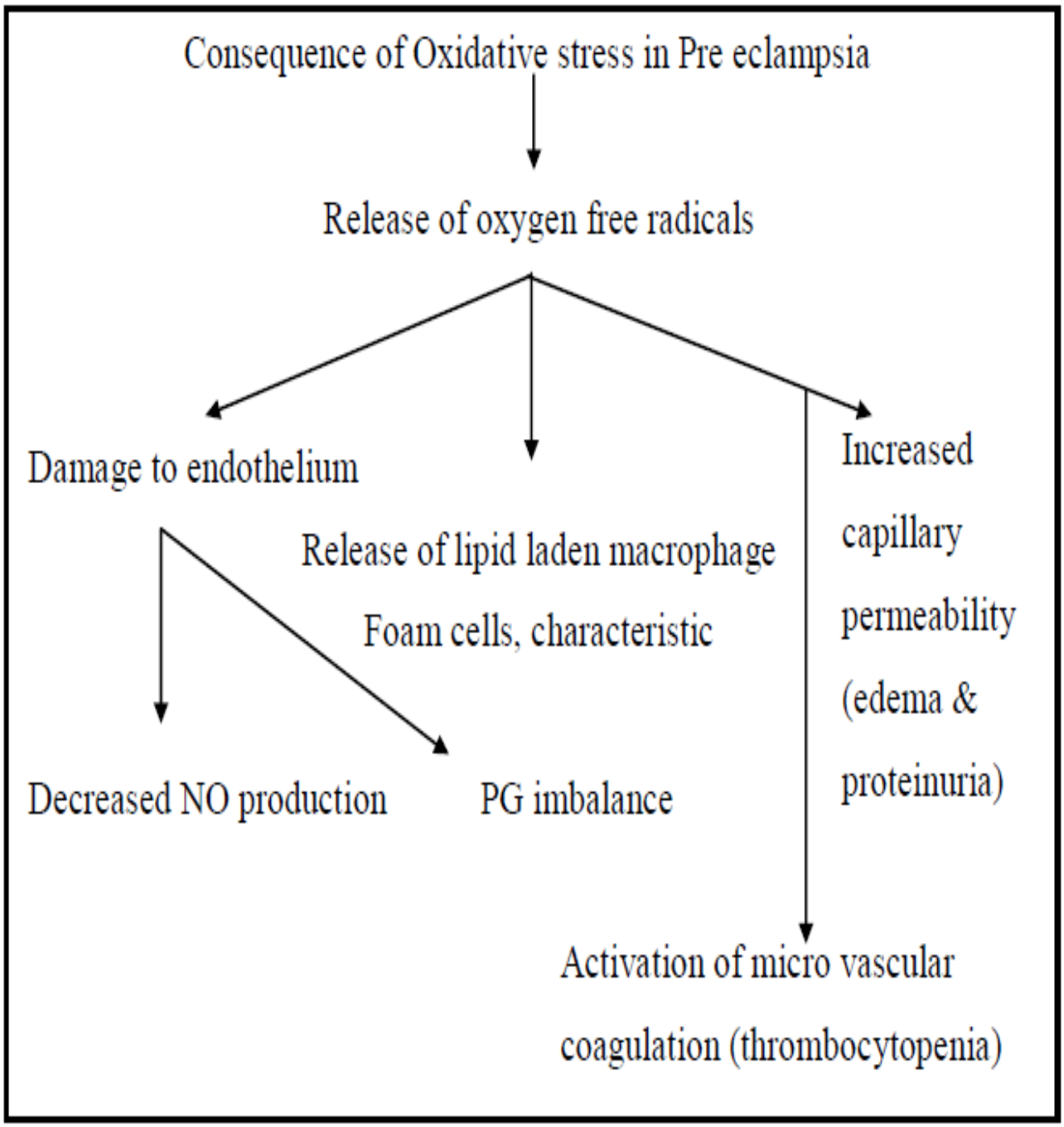
Nitric Oxide acts along with prostacyclin which is a potent vasodilator and platelet aggregator. In preeclampsia there is an imbalance between prostacyclin and thromboxane and decreased synthesis of nitric acid leading to vasoconstriction [65-66].

OXIDATIVE STRESS:

At present themost accepted theory is "OXIDATIVE STRESS ". Reduced placental perfusion leads to increased levels of prooxidants over antioxidants and release of free radicals. This imbalance between antioxidants and prooxidants results in oxidative stress which leads to increased TXA2 levels and decreased PGI2 levels and causes endothelial dysfunction [67].

In preeclampsia there is decreased expression of thioredoxin and glutaredoxin in the placenta which leads to greater accumulation of modified renal proteins which are the markers for lipid per oxidation in the placenta of preeclampsia.

There occurs increased production of nitric oxide in preeclampsia as a compensatory process which competes with superoxide dismutase and combines with superoxide.



VASOSPASM:

VOLHARD et al states that the hallmark of preeclampsia is vasospasm. Vasospasm leads to endothelial damage and interendothelial cell leakage leads to deposition of fibrin and platelets in the sub endothelium and causes uteroplacentalinsufficiency.

INCREASED PRESSOR RESPONSE:

Normally during pregnancy there occurs refractoriness to the pressor effects of Angiotensin II. For the maintenance of normal vascular refractoriness an adequate balance between the production and metabolism of vasoactive prostaglandins is responsible. In preeclampsia when compared to normal pregnancy this ratio is altered and there is decrease in the production of PGE2 & PGI2 and increased production of TXA2 which leads to increased sensitivity of vasoactive amines causing vasoconstriction.

ABNORMAL TROPHOBLASTIC INVASION:

During pregnancy there occurs a physiological change in the spiral arterioles which convert them into uteroplacental arteries which occurs in two stages. During the first stage there occurs endovascular trophoblastic invasion of the decidual segment of the spiral arterioles. During 16 to 20 weeks of gestation there occurs secondary trophoblastic invasion of the myometrial segment of spiral arterioles which converts them into more elastic tortuous easily distensible channels.

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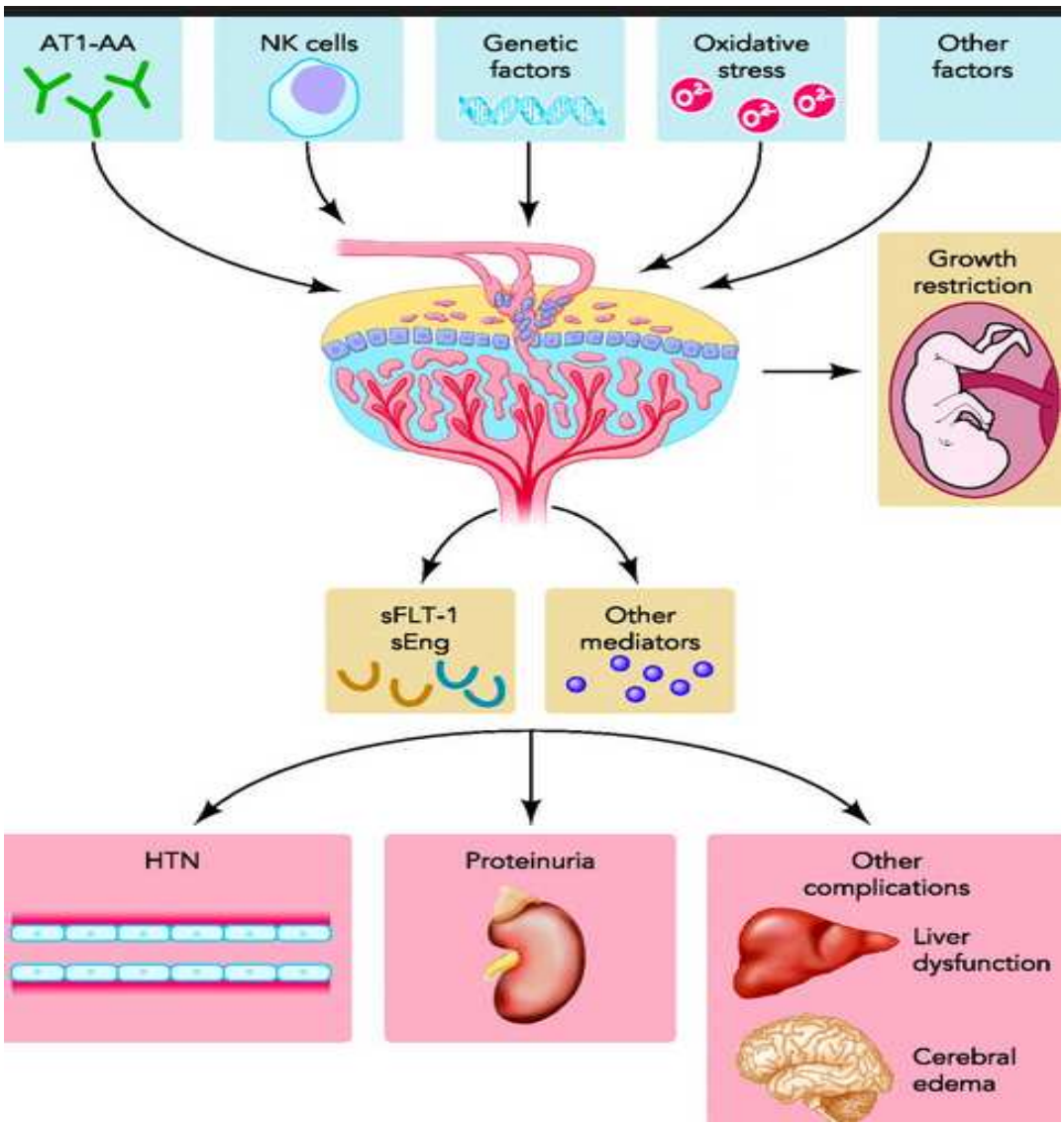
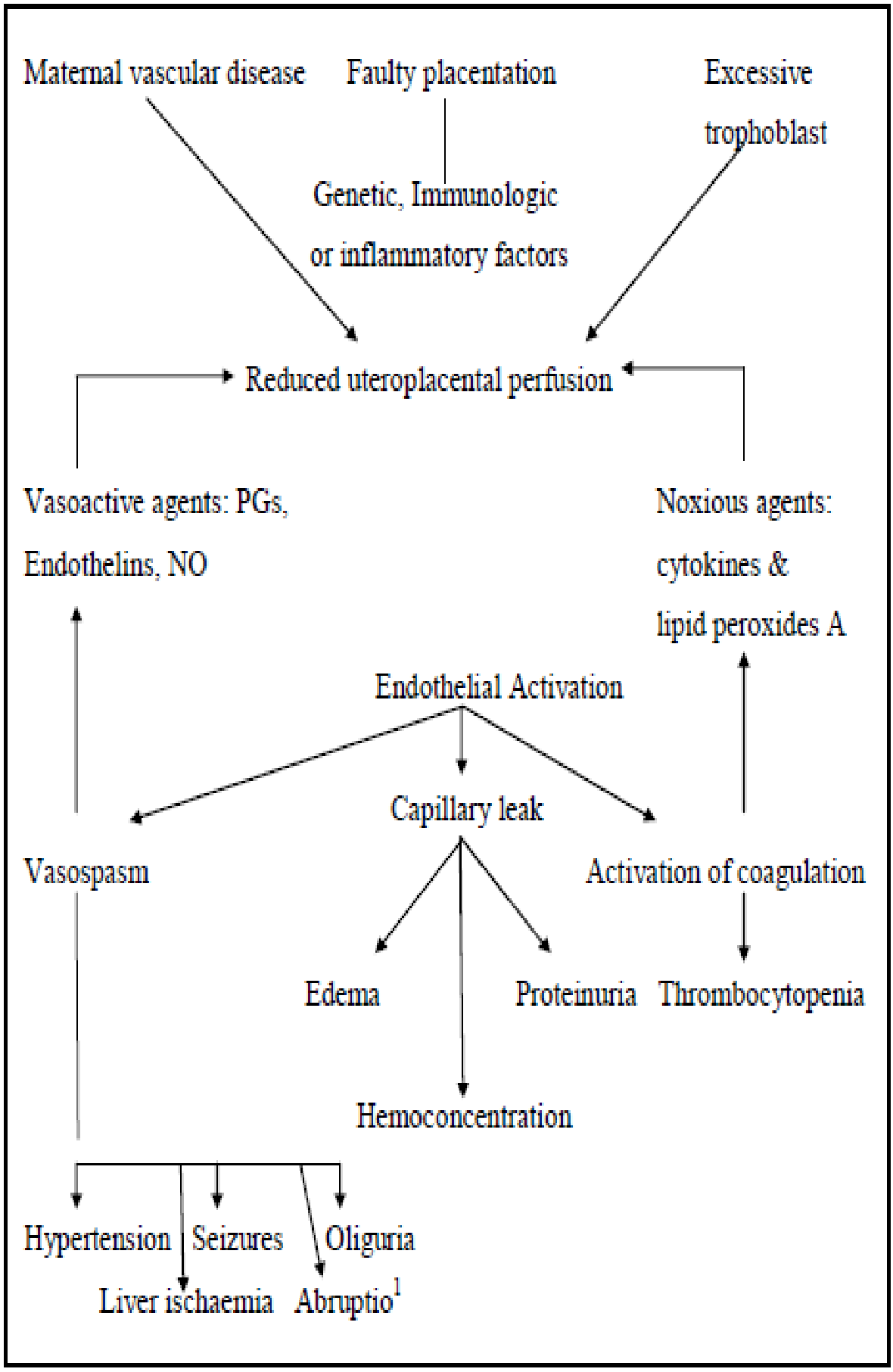


FIGURE:2: Demonstrates The Pathophysiology Of Preeclampsia And Its Consequences.

PATHOPHYSIOLOGICAL CHANGES:

HAEMODYNAMIC CHANGES:

- In preeclampsia there occurs generalised vasospasm in contrast to normal pregnancy which shows hypervolemia and hence there is decrease in preload.
- Due to increase in vascular resistance there is increase in afterload.Increased afterload causes decreased cardiac output.
- Due to increase in cardiac output and vascular resistance MAP increases.
- Haemoconcentration is the hallmark of preeclampsia.
- Reduced circulating blood volume in women homozygous for T235 angiotensin gene type associated with preeclampsia. (Silver and Associates 2001).
- Increased sensitivity to Angiotensin II.
- Increased peripheral resistance.
- Increased blood pressure.
- Intrinsic renal changes.
- Reduced renal blood flow.
- Reduced glomerular function.


Maternal vascular disease
Faulty placentation
Excessive trophoblast
Genetic, Immunologic or inflammatory factors
Reduced uteroplacental perfusion
Vasoactive agents: PGs, Endothelins, NO
Noxious agents: cytokines & lipid peroxides A
Endothelial Activation
Capillary leak
Vasospasm
Activation of coagulation
Edema
Proteinuria
Thrombocytopenia
Hemoconcentration
Hypertension
Seizures
Oliguria
Liver ischaemia
Abruptio[1]

HAEMATOLOGICAL CHANGES:

THROMBOCYTOPENIA:

It occurs in 10 -20 % of cases of preeclampsia. It occurs as an immunologically mediated process due to increased platelet deposition at the site of endothelial damage. The severity of thrombocytopenia is directly proportional to the maternal and fetal morbidity and mortality.

COAGULATION SYSTEM:

The presence of fibrin and thrombin in the microvasculature of various organs suggests that preeclampsia is a hypercoagulable state. The vasospasm which is characteristic of preeclampsia causes endothelial injury which favour fibrin deposition in microvasculature.

INCREASED	DECREASED
Activity of intrinsic pathway factors↑FV IIIa: FV IIIc activity Thrombin : Antithrombin ratio ↑Platelet aggregation	Antithrombin III Fibrinogen

CARDIOVASCULAR CHANGES:

- Sensitivity to Angiotensin II is increased.
- Haemoconcentration and decreased circulatory blood volume.
- Increase in heart rate and hyper dynamic ventricular function.
- Increase in peripheral resistance.

RENAL SYSTEM:

- Reduced renal blood flow and glomerular function.
- Intrinsic renal changes.
- Reduced uric acid clearance
- Proteinuria Glomerular capillary endotheliosis.

ACUTE RENAL FAILURE:

Acute tubular necrosis is rarely associated with isolated preeclampsia. When preeclampsia is associated with either abruption or HELLP there is more probability of acute tubular necrosis. Irreversible cortical necrosis develops rarely.

HEPATIC SYSTEM:

Involvement of hepatic system was first described by VIRCHOW in 1856.In the periphery of the liver there are regions of periportal haemorrhage. Liver

involvement associated with haemolysis, thrombocytopenia and elevated liver enzymes is popularly termed as HELLP.

Clinically manifests as epigastric pain or tenderness in the upper epigastrium and are associated with elevated AST & ALT.

Sometimes presents as asymptomatic elevations of AST & ALT and platelet count which normalises following delivery within three days.

Infarction in the liver can lead to hepatic haemorrhage which inturn leads to hepatic haematoma causing subcapsular haemorrhage. MRI & CT are used in the diagnosis .Most of them are treated conservatively however surgical intervention is life saving in few cases.

Women with HELLP syndrome have increased risk of associated complications like Eclampsia, Abruptio placenta, pulmonary edema, coagulopathy, Acute renal failure, compared to women with isolated preeclampsia.

BRAIN:

Visual symptoms and headache are commly associated with severe preeclampsia. When it is associated with convulsions it is termed as eclampsia. Cerebral involvement can be identified by MRI,CT,Doppler studies.

PATHOPHYSIOLOGY:

There are two theories which explains the pathophysiological changes,

In response to acute or severe hypertension

⇩

Cerebral overregulation

⇩

Vasospasm

⇩

Ischemia

⇩

Cytotoxic edema

⇩

Infarction

sudden increase in blood pressure

⇩

Exceeds normal cerebral auto regulation at capillary level

⇩

Increased hydrostatic pressure, hyper perfusionextravasations of plasma ,

⇩

Vasogenic edema

CLINICAL FEATURES:

- Headache and Scotomas: Occurs due to cerebral hyper perfusion more in the occipital lobe.
- Convulsions: Are diagnostic of eclamps.
- BLINDNESS occurs when preeclampsia is associated with eclampsia.

- Cerebral edema leads to altered mental status , confusion and coma. Sometimes can lead to fatal supratentorial herniation.

VISUAL CHANGES:

Blurringof vision,scotoma, Diplopia are associated with severe preeclampsia. Blindness when occurs is usually reversible occurs due to involvement of retina, occipital lobe,and lateral geniculate body.

Purtscher retinopathy is the term used to describe the blindness that occurs due to retinal ischemia or infarction. Altered vision can also occur due to retinal detachment which is mostly unilateral.

UTEROPLACENTAL INSUFFICIENCY:

Vasospasm leading to compromised uteroplacental perfusion contributes to the perinatal morbidity and mortality. Uterine artery blood flow measurement estimates uteroplacental perfusion. With abnormal placentation high resistance persists in the uterine artery. Persistant uterine artery notching is associated with increased risk of IUGR or preeclampsia.

PREDICTORS OF PREECLAMPSIA:

There are tests which can predict the occurance of preeclampsia. The various tests are

- Tests depending on blood pressure measurement.
- Urine assay.
- Blood assay.
- Angiotensin I sensitivity test.

TESTS DEPENDING ON BLOOD PRESSURE MEASUREMENT:

ROLL OVER TEST:

The patient is asked to lie in lateral decumbent position and blood pressure is measured, then after 5 minutes patient is asked to lie supine and blood pressure is recorded again. An increase in blood pressure reading > mmHg

MID TRIMESTER MEAN BLOOD PRESSURE:

Mean arterial blood pressure >90 mmHg has more than four fold increase in risk of preeclampsia.

HAND GRIP TEST:

Isometric sustained contraction of striated muscle generates sympathetic activity increases arterial blood pressure. The patient compresses an inflated sphygmomanometer cuff at its maximumfor 3 minutes and then at 50% of maximum. An increase in DBP >20mmHg at 28-30 wks increases the risk.

URINE ASSAY

- Microalbuminuria**:**Detected by radioimmunoassay before albuminuria can be detected by ordinary methods.
- 24 Hrs Urine Calcium: excretion is decreased in women with preeclampsia than normal women.
- Kallikerin Vs Creatinine: Ratio decreases in patients who are prone for preeclampsia.

BLOOD ASSAYS:

- PLASMA URATE LEVELS serial increase is a warning of PIH before appearance of other clinical features.
- Reduced platelet count occurs early in preeclampsia.
- Antithrombin III activity begins to decline as much as 13 wks prior to the development of manifestations of preeclampsia.

ANGIOTENSIN II SENSITIVITY TESTS:

- Sensitivity to infused angiotensin II is increased due to alteration in the vascular smooth muscle AT II receptors.
- Platelet and angiotensin binding is increased before development of preeclampsia.

COMPLICATIONS OF HYPERTENSIVE DISORDERS OF PREGNANCY:

MATERNAL:

- Eclampsia.
- Abruptio placenta.
- HELLP Syndrome.
- Rupture of liver.
- Pulmonary Edema.
- Cerebral Haemorrhage.
- Retinal Detachment.

FETAL COMPLICATIONS:

- Prematurity.
- IUGR.
- Oligohydramnios.

- ICH.
- Respiratory Distress.
- Magso4 to mother causes neonatal depression.

PROTEINURIA

Proteinuria is defined as urinary protein excretion of>150 mg/ day in a 24 hr urine collection. It denotes an increased glomerular permeability of the plasma macroglobulin albumin which is normally not filtered in the urine.

Normally urine protein excretion per day is < 150 mg of which albumin constitutes 60% and tubular Tam Horsfall protein 40%.

Proteinuria in pregnancy is defined as an urinary protein excretion of > 300mg/ day in a 24 hr urine collection. Incidence of proteinuria in pregnancy is 5% [(68)].

Though proteinuria indicates the severity of the disease absence of it does not exclude the severity of preeclampsia. There are cases where preeclampsia and eclampsia occur without proteinuria [(69)].

TYPES OF PROTEINURIA

GLOMERULAR PROTEINURIA:

It is characterised by proteinuria due to damage to the basement membrane. It occurs in glomerular disease and is also termed as albuminuria.The glomerulus which behaves as an ultra filterfor plasma proteins is composed of endothelial cells, basement membrane and epithelial foot processes. The filtration process depends upon

MOLECULAR SIZE:

There is an inverse relationship between the size of the particle and glomerular filtration. IgM which is a HMW protein cannot undergo filtration whereas albumin a LMW protein can easily pass through the filter.

CHARGE:

Endothelial cells and the basement membrane normally posses a negative charge which repels the negatively charged proteins like plasma albumin.

CONCENTRATION IN PLASMA:

Higher the plasma concentration the more it appears in the urine, hence albumin appears in the urine when compared to low concentration proteins.

RESORPTION BY RENAL TUBULES:

Albumin is less absorbed when compared to VLMW Proteins and hence about 60% appears in urine. In selective proteinuria LMW proteins are filtered whereas in non selective proteinuria the filtration is independent of the molecular weight.

FUNCTIONAL PROTEINURIA:

It is related to alterations in blood flow throughout the glomeruli. Whenever there is excessive blood flow like fever, exercise and increased stress there

occurs functional proteinuria with protein excretion <1 gm/day.The condition becomes normal once the precipitating event is withdrawn.

POSTURAL /ORTHOSTATIC HYPERTENSION:

Increase in proteinuria with upright position is called postural proteinuria with excretion >1 gm/day. Whenpersistent signifies renal pathology.

TUBULAR PROTEINURIA:

It occurs due to tubular or interstitial injury which impairs the reabsorption of Low Molecular Weight protein and hence they appear in urine[70].

OVERLOAD PROTEINURIA:

It occurs when the reabsorptive capacity is impaired as a result of increased production of protein and the excess protein is filtered in the urine.

MICROALBUMINURIA:

Excretion of >30mg and <300mg/ day of albumin. It is the earliest evidence of type 1 diabetes.

KIDNEYS IN NON PREGNANT WOMEN:

The upper limit of protein excretion is 150 mg /dl of which 40% forms albumin and 60% Tomm Horsfall protein.

There are various factors like size, charge, molecular weight which influences the movement of protein across the glomerular capillary bed. The proximal tubular epithelial cells absorbs the protein filtered by the glomeruli by endocytosis,but this process is saturated with high glomerular protein load and causes proteinuria.

KIDNEY IN UNCOMPLICATED PREGNANCY:

There are both anatomical and physiological alterations in kidney during pregnancy. The kidneys increase in size and there is enlargement of renal calyx, renal pelvises and ureters enlarge more commonly on the right side under both hormonal influence and obstruction. There is increase in renal plasma flow and glomerular filtration rate. There is increase in creatine clearance which causes decreased serum Creatinine levels.

PROTEINURIA AND PREECLAMPSIA:

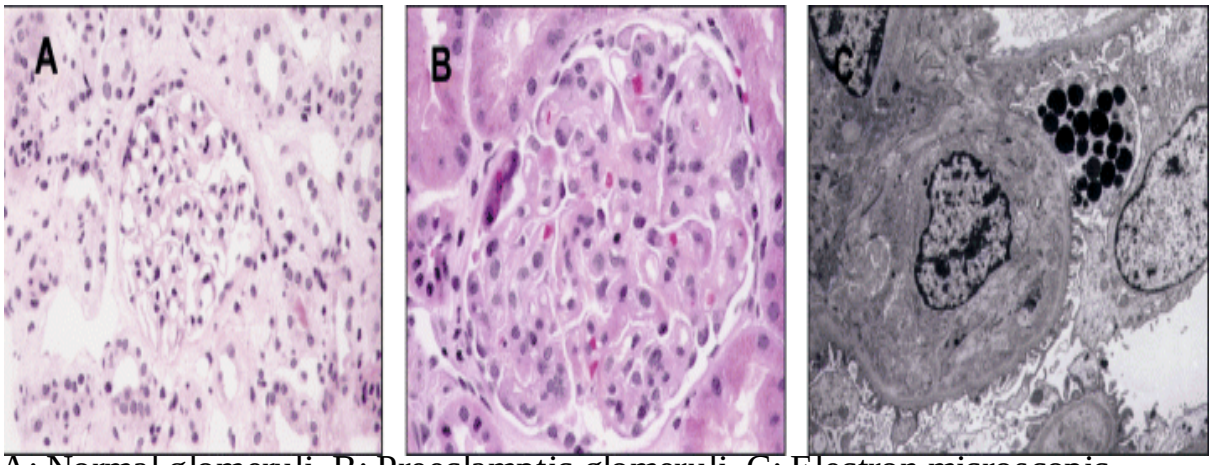
Proteinuria forms an essential criteria for the diagnosis of preeclampsia. The presence of proteinuria can lead to many pregnancy complications from urinary tract infections to chronic renal disease. It is the hallmark of preeclampsia and the degree of proteinuria is a sign of worsening of hypertensive disorders.

KIDNEY CHANGES IN PREECLAMPSIA:

Both morphological and functional characteristics of kidney are affected in preeclampsia. Since the glomerulus is affected there is excretion of albumin in the urine. Preeclamptic glomeruli undergo structural changes with hypertrophy of cytoplasmic organelles and endothelial vacuolisation as defined as glomerular Endotheliosis.

In preeclampsia due to hypertrophy and proliferation of intercapillary cells the glomeruli is bloodless and diffusely enlarged. Ultra structurally it is described as hypertrophy of cytoplasmic organelle in endothelium leading to marked enlargement and vacuolisation as a result of accumulation of free lipids. This is classically described as glomerulo capillary Endotheliosis .

Preeclampsia can rarely cause acute renal failure which is due to tubular necrosis. When a major portion of cortex is damaged renal cortical necrosis occurs which is irreversible.



A: Normal glomeruli, B: Preeclamptic glomeruli, C: Electron microscopic picture of preeclamptic Glomeruli

FIGURE: 3: Glomerulocapillary Endotheliosis

PATHOPHYSIOLOGY OF PROTEINURIA IN PREECLAMPSIA:

Spasm of glomerular arteries

⇩

Anoxia of glomeruli

⇩

Endothelial Damage

⇩

Escape of proteins into urine

PROTEINURIA AND CLINICAL OUTCOME:

Significant proteinuria >300 mg/dl or > 2+ dipstick is associated with increase in perinatal morbidity like fetal growth restriction, stiilbirth, preterm deliveries before 37 wks and increased neonatal death. Even the trace proteinuria is associated with poor outcomes.

Ginsberg et al showed that the degree of proteinuria in both diagnostic and prognostic significance.

Outcome of 400 pregnancies complicated by proteinuria and hypertension are studied and found a four times of risk of perinatal morbidity and mortality in women with proteinuria than in non proteinuric group

FERRAZZANI et al studied 444 hypertensive women with proteinuria and noted increase in levels of serum uric acid ,low birth weight and more deliveries before 37 completed weeks.

Sudan et al showed that complications increases with the degree of proteinuria.

In a prospective study by SCHIFF et al showed that in patients with proteinuria there is increased perinatal morbidity in terms of IUGR, LBW, NICU admissions.

DIAGNOSIS:

In all hospitals whether it is a primary health centre or a private hospital all antenatal women at their booking visit should be screened for proteinuria and also at subsequent visits. If the dipstick shows proteinuria it has to be evaluated [72].

DIPSTICK TEST:

A Dipstick contains reagent for one or more tests for example ketone bodies , proteins and glucose[73]. In the presence of proteins there is a colour change in the reactive portion of stick. The dipstick is buffered with citrate PH3 buffer and tetrabromophenol .In the presence of protein blue turns yellow. Since the first morning sample is more concentrated and less affected by postural factors it is usually preffered for detection of proteinuria.

Due to alkaline and concentrated urine and also due to contamination by vaginal discharge there are large number of false positives [74] . Dilute urine may sometimes produce false negative reaction.

Sudan et al showed that the high number of false negatives with urine Dipstick when compared to 24 hr urine measurement.

Meyer et al found that Dipstick is a poor predictor of absent or severe proteinuria.

Jasone et al showed Dipstick values of trace or 1+ correlates poorly whereas at higher degrees it correlates better.

TRACE	15mg/dl	
1+	30mg/dl	<0.5 gm/ day
2+	1oomg/dl	0.5 -1 gm/ day

3+	3oomg/dl	1 -2 gm/ day
4+	2gm/dl	> 2gm /day

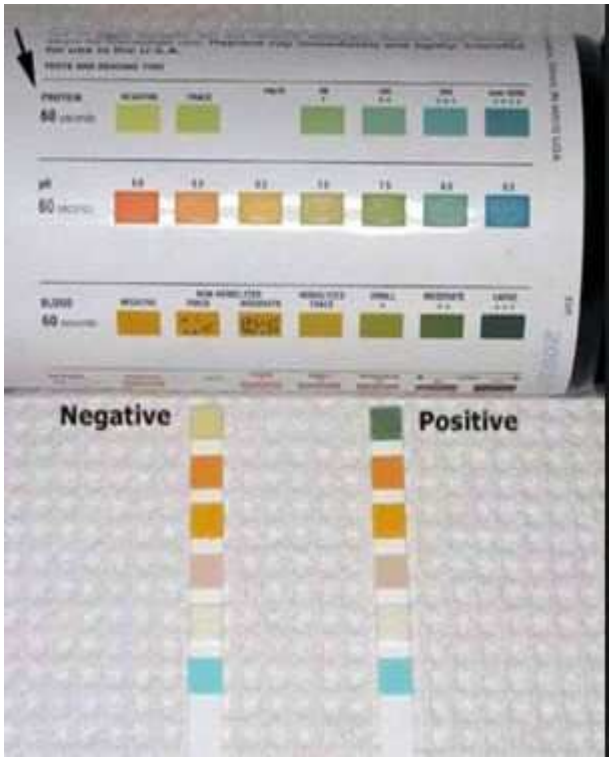


FIGURE 4 : Urine Dipstick And Interpretation

SULFOSALICYLIC ACID TEST:

Detects protein by acid precipitation and any time of protein can be detected.Three parts of 3% sulfosalicylic acid is mixed with one part of urine and the amount of protein is quantified by an auto analyser.

HEAT COAGULATION TEST:

It is done by adding acetic acid 3 – 4 drops to urine after heating.

Depending upon the turbidity the results are graded. False positives and false negative results can occur.

QUANTIFICATION OF PROTEIN:

Quantification is important to differentiate proteinuria caused by various renal diseases. Tubulo interstitial diseases cause proteinuria of < 1 – 2 gm / day and glomerular disease > 3.5 gm/ day. Quantification is usually done by timed collections of which 24 HRS urine sample remains the gold standard.

24 HOURS COLLECTION:

It is the GOLD STANDARD for quantification of proteinuria. Protein excretion is variable in the setting of preeclampsia therefore it is recommended that 24 hr urine collection is used for the diagnosis .The combination of hypertension and diabetes during pregnancy markedly increases perinatal morbidity and mortality. Proteinuria is a sign of worsening of hypertensive disease and predicts the outcome of preeclampsia. Based on the degree of Proteinuria the management decision varies .Overestimation of Proteinuria leads to interventions that may end in preterm delivery whereas underestimation leads to delay in the diagnosis of severe nephropathy leading to kidney damage.

Throughout the day there is variation in the rate of protein excretion which is secondary to vascular spasm and vasoconstriction producing fluctuation in protein excretion. There is an increase in Proteinuria in an upright positionand

ambulation causing vasoconstriction and increases glomerular permeability. These changes produces a diurnal variation in protein excretion. There occurs a circadian rhythm in the excretion of potein so there is a necessity for a 24hr urine collection for quantification of Proteinuria. In hospitalised patients there is not of much variation in the circadian rhythm and it is therefore possible to use shorter collection specimen.

Although accurate 24 hr collection is time consuming and cumbersome and requires patient compliance hence it can be replaced by a shorter collection which gives a similar result. Kieler H, Zettergran T, Svensson H Showed that the goldstandad 24 hrs urine excretion for assessment of Proteinuria in preeclamptic women can be substituted by 12 hr urine collection .Study conducted by Haemak medical centre Israel comparing 24 hrs urine protein vs shorter period on 300 patients and found significant correlation between the two.Mayo Clinic conducted a study which showed excellent correlation between 12 hrs and 24 hrs.

Sherry Boschart et al during the management of preeclampsia instead of 24 hrs urine collection practiced 12 hrs urine collection Spot urine samples are inaccurate and therefore not recommended for quantification of Proteinuria . Minoo Rajaei et al studied 63 pregnant women and compared 24 hrs urine protein with 12 hrs urine protein and found that significant correlation with

sensitivity of 83% and specificity of 91%, positive predictive value of 80% & negative predictive value of 82%, pearson's correlate was 0.890.

Some investigators explored methods for quantifying proteinuria in a shorter period. The PCR of a single voided urine sample correlated with 24 hrs when proteinuria is < 1gm/day and poor correlation when proteinuria is > 2gm /day. Christina Tun MD et al studied 102 patients and compared spot PCR, 12 hrs urine protein and 24 hrs urine protein. Both 12 hrs and 24 hrs urine protein correlated well with 24 hrs urine protein.Jonne N Quinones et al studied 90 women comparing spot PCR, 12 hrs and 24 hrs urine protein correlated better than PCR with sensitivity 96%& 89%, specificity 100%& 40% , positive predictive value 100% & 32% and negative predictive value 98% & 91% respectively

Methods comparing 2 hrs, 8 hrs specimens are also studied. 2 hr specimen correlated with no proteinuria but does not correlate in cases of severe proteinuria. The need for 24 hrs collection is because of high degree of variation in the urine protein excretion throughout the day and the factors influencing are

- Variation in water intake and excretion.
- Rate of dieresis.
- Diet.
- Posture.
- Exercise.

So a single sample spot PCR cannot accurately predict proteinuria and hence the need for shorter collection specimen which will give accurate result like 24 hrs specimen is appropriate. Our study is to find whether 12 hr collection correlates with 24 hrs collection.

Sogra Rabiae et al studied 57 patients and showed 12 hr sample correlated with 24 hrs sample for patients with no disease ($p < 0.02$) mild proteinuria ($p<0.02$), sevsre proteinuria ($p < 0.01$).

.Irruae specialist teaching hospital issue compared 2 hr, 12 hr, & 24 hr urine specimen and found a $P < 0.02$ and sensitivity of 96%, specificity of 98% and Negative predictive value of 96%.

F Tare, A Monsouri, F Ravanbaksh showed that shorter urine collection showed better correlation equal to 24 hr collection.

Tun C, Quinones JN ,Kurt A Studied and showed that higher correlation of 12hr urine protein >165 correlated with a 24 hr urine protein >300 mg with the benefit of a shorter evaluation time for Proteinuria.

Adelberg AM, Miller J, Doezbacher M Studied 65 hypertensive women and showed that total protein values of 8 & 12 HRS correlate positively with values of 24 hrs sample for patients with Proteinuria. The results of 12 and 24 hrs coincides in cases even without Proteinuria.

III. AIM OF THE STUDY

The objective of the study was to know if 12h hrs urine protein would provide an accurate quantification of proteinuria and whether it can replace the use of 24 hrs urine protein in hypertensive disorders of pregnancy

IV. MATERIALS AND METHODS

This study was conducted at the department of obstetrics and gynaecology, Govt R.S.R.M Lying in hospital attached to Stanley Medical college, Royapuram ,Chennai.

STUDY PERIOD:

The study period was from August 2012 –July 2013.

STUDY DESIGN:

A total of 100 pregnant women > 20 Weeks gestation with preeclampsia and gestational hypertension attending Government RSRM Lying in Hospital attached to STANLEY MEDICAL COLLEGE Chennai will be selected consequently as and when they are presented with following inclusion and exclusion criteria.

INCLUSION CRITERIA:

- Pregnant women with preeclampsia or gestational hypertension with blood pressure≥140/90mm Hg or more, recorded on two occasions 6 hrs apart.
- Single diastolic blood pressure recording of ≥110mm Hg.
- Presence of proteinuria of trace or more detected by a test done on a random urine sample.

EXCLUSION CRITERIA:

- Chronic hypertension
- Preexisting renal disease
- Gestational Diabetes Mellitus.

METHODOLOGY:

Those patients who satisfy the above criteria are to be selected for the study.

The test has to be carried out in hospitalized patients.

- A detailed history has to be taken.
- General physical examination.
- Systemic examination.
- Obstetric examination.

A qualitative urine test for urine protein has to be done on a random urine sample using dipstick method and graduated as follows

TRACE 15mg/dl

1+	30mg/dl<0.5 gm/ day	
2+	1oomg/dl	0.5 -1 gm/ day
3+	3oomg/dl	1 -2 gm/ day
4+	2gm/dl> 2 gm / day	

The test has to be repeated and if the repeat test shows trace or above quantitative estimation for proteinuria has to be carried out as follows

The patient should be instructed to collect the 24 hr urine specimen from the second urine sample as two consecutive 12 hr collections in separate containers.The containers should be labeled with the patients name, age, IP NO. For emergency purposes sample can be collected at any time of day. At the hour selected to start the patient have to urinate and discard the sample and start collecting from the next sample. Collect all the urine passed in a clean container and immediately transfer to the collection bottle .Have the patient pass the last urine at the end of 24 hr period and add into the bottle .The container must b kept in the refrigerator at all times during the 24 hrs collection.

The following factors affect the test sample

- Lack of fluid (Dehydration)
- Vaginal secretion
- Severe emotional stress.

- Strenuous exercise.
- Urinary tract infection.

The best way to avoid retention error is to have the patient well hydrated and positioned in lateral recumbency position before starting and finishing the collection which minimizes the dead space errors and ensure that any urine in the urinary tract or bladder is of recent origin.

The total urine volume collected is measured by graduated cylinders and send to the laboratory as early as possible and total protein were measured by pyrogallol Red Molybdate method.6ml of 12 hrs sample was taken as the first sample, the remaining first 12 hr sample is mixed with the second 12 hour sample and 6ml of the result solution was considered as second sample. Proteinuria is defined as >150mg in 12hours and >300mg in 24 hours.

NORMAL VALUES OF PROTEIN EXCRETION [93]:

In the 24 hrs urine protein estimation upto 300mg is assigned as no proteinuria, mild proteinuria is 300 -3000 mg and severe proteinuria is > 3000mg.in the 12 hrs urine protein estimation < 165mg is assigned as no proteinuria ,165 -1650 mg is considered as mild proteinuria and >1650 mg is considered as severe proteinuria.[9]

	12 hrs urine protein (mg/dl)	24 hrs urine protein (mg/dl)
NO PROTEINURIA	<165	<300
MILD PROTEINURIA	165-1650	300 -3000
SEVERE PROTEINURIA	>1650	>3000

Semi Auto Analyser With Microproteinuria Kit Used For The Study:

METHODS:

The urine protein concentration is measured in the laboratory by pyrogallol red molybdate method.

- Measure the 24 hrs urine volume in ml.
- Divide the volume by 100 to determine the volume expressed in decilitre.
- Multiply the 24 hrs urine volume in dl by the protein concentration in mg/dl as determined by Pyrogallol Red Method,[96]
- The resulting number is the amount of protein expressed as mg/dl.

V. STATISTICAL ANALYSIS

The data thus collected were analyzed using appropriate statistical methods. The mean and standard deviations were computed. The statistical tests used for analyses were the

- Pearson correlation coefficient which is expressed as r.
- Student Chi square test expressed as p value of $p < 0.05$ has been considered to be statistically significant**.**

Table 1: Demography**:**

	N	Minimum	Maximum	Mean	Std. Deviation
Age in years	100	17	40	23.87	4.228
Gestational Age	100	24	38	35.16	3.719
SBP	100	130	180	149.10	9.650
DBP	100	90	110	94.10	5.877
12Hrs Urine Protein	100	40	2280	543.52	656.107
24Hrs Urine Protein	100	88	4450	1044.61	1262.626
Valid N (list wise)	100				

This tabulation demonstrates the various variables dealt in the study their distribution, mean, standard deviation. In the present study it is observedthat the mean $\pm$SD of age of the subject studied was 23.87 ± 4.22, gestational age was

35.16± 3.719, SBP was 149.1± 9.65, DBP was 94.1± 5.877, 12 hrs urine protein was 543.5± 656.1, 24 hrs urine protein was 1044.6± 1262.6.

Table 2: Age wise distribution of cases:

Age	No.of pts	Percentage %
<21	45	45%
21-28	43	43%
>28	12	12%
Total	100	100%

MacGillivary (1958) showed a J shaped curve between the relationship of maternal age and incidence of preeclampsia with high incidence among young and elderly age groups. In our study also there is a higher incidence among age groups 17-21 (45%) and >28 yrs(12%) together they make up 57%. In our study the minimum age of the patient is 17 yrs and maximum age is 40 yrs.

Chart 1: Age wise distribution of cases:

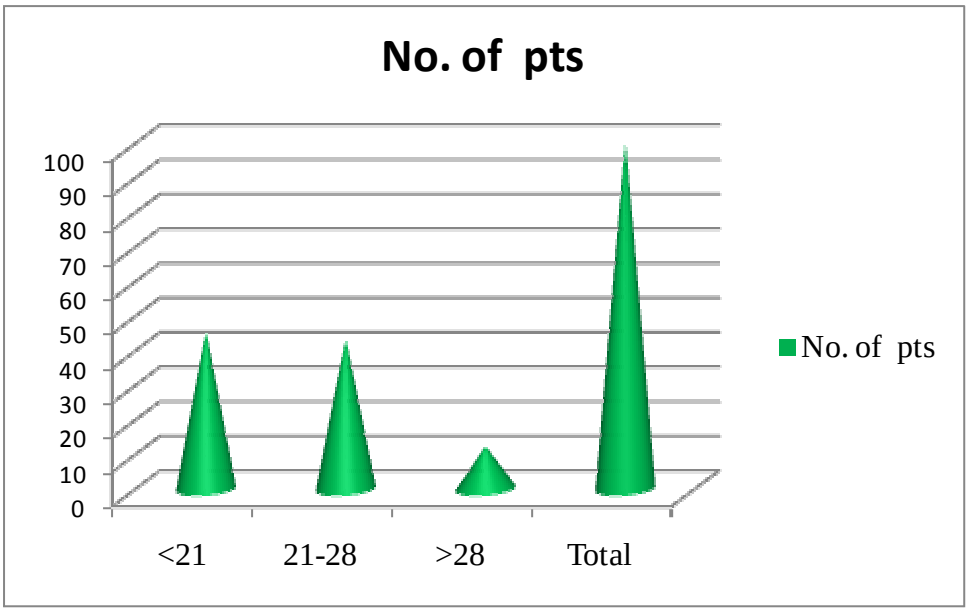


Table 3: Age wise distribution of cases according to the degree of Proteinuria:

		12Hrs Urine protein			24Hrs Urine Protein		
		No Proteinuria	Mild proteinuria	Severe Proteinuria	No Proteinuria	Mild proteinuria	Severe Proteinuria
Age in years	17-21	21	18	6	20	19	6
	22-78	8	28	7	7	28	8
	>28	5	5	2	5	5	2

In our study there is an excellent correlation between 12 hrs urine protein and 24 hrs urine protein among various age groups and various degrees of proteinuria. This table shows that among the 34 cases who had no proteinuria in the 12hrs group 32 cases had no proteinuria 2 cases had mild proteinuria. Among the 51 cases who had mild proteinuria in the 12 hrs group all had mild proteinuria even in the 24 hrs group. Among the 15 cases who had severe proteinuria in the 12 hr group all had severe preeclampsia and one patient who had mild proteinuria in the 12 hr group .

Chart 2: Age wise distribution of cases according to the proteinuria:

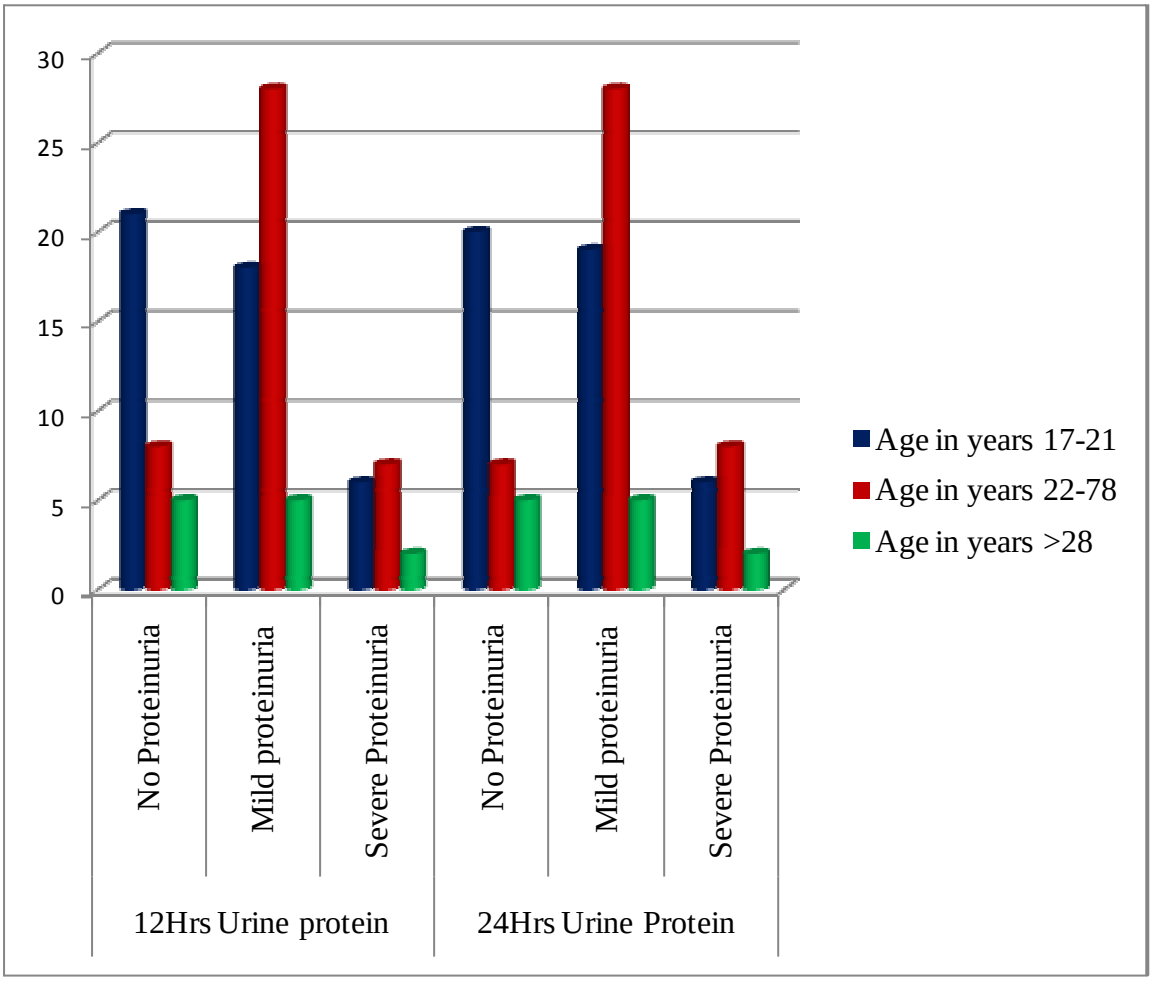


Table: 4: Parity wise distribution of Cases:

Parity	No.of pts
Primi	66
Multi	34

The above table and chart shows the distribution of cases with preeclampsia according to parity. According to various theories preeclampsia is more common in nulliparous women. In our study among the 100 cases 66 are nulliparous and 34 are multiparous with a P value < 0.001 which is statistically highly significant.

Chart 3: Parity wise distribution of Cases:

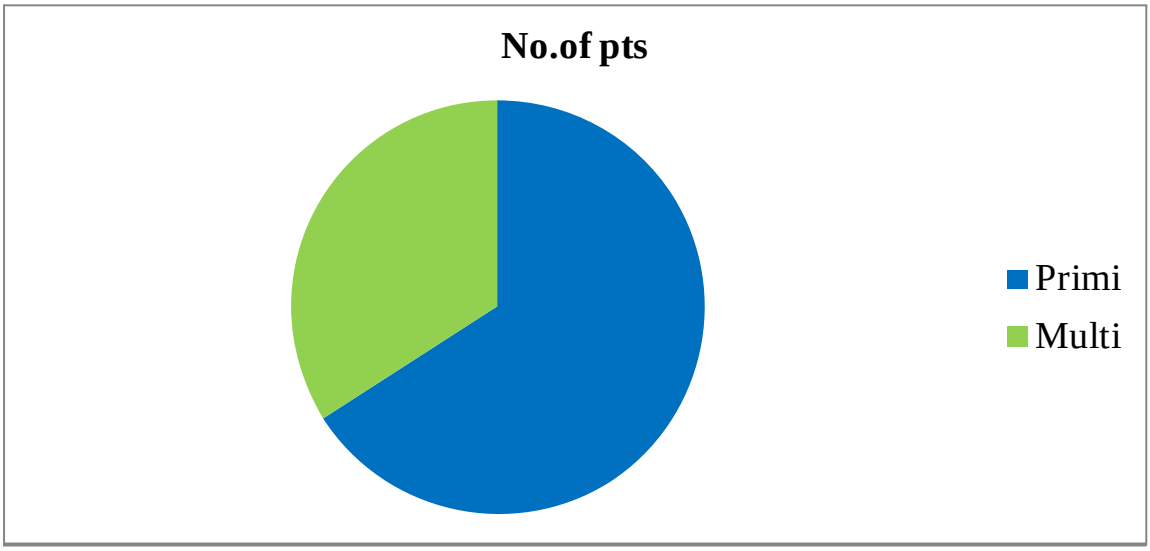


Table 5: Parity wise distribution of cases according to the degree of Proteinuria:

		Primi	Multi
12Hrs Urine protein	No priteinuria	22	11
	Mild proteinuria	35	17
	Severe proteinuria	7	6
24Hrs Urine protein	No priteinuria	22	10
	Mild proteinuria	34	18
	Severe proteinuria	10	6

This table demonstrates the distribution of various degrees of proteinuria among primigravida and multigravida. There is no difference in the distribution of various degrees of proteinuria and gravida in both 12/24 hrs urine protein sample. Hypertensive disorders of pregnancy were common in primi. Sibai& colleagues (1993) confirmed the higher risk of developing hypertension in the first pregnancy. According to a study conducted in cape peninsula (1979-81) there was double the incidence of hypertension & proteinuria in primi.

In our study primi constitutes 66% with a p value of <0.001which is statistically significant, it correlates with the studies.

Chart 4: Parity wise distribution of cases according to the degree of Proteinuria:

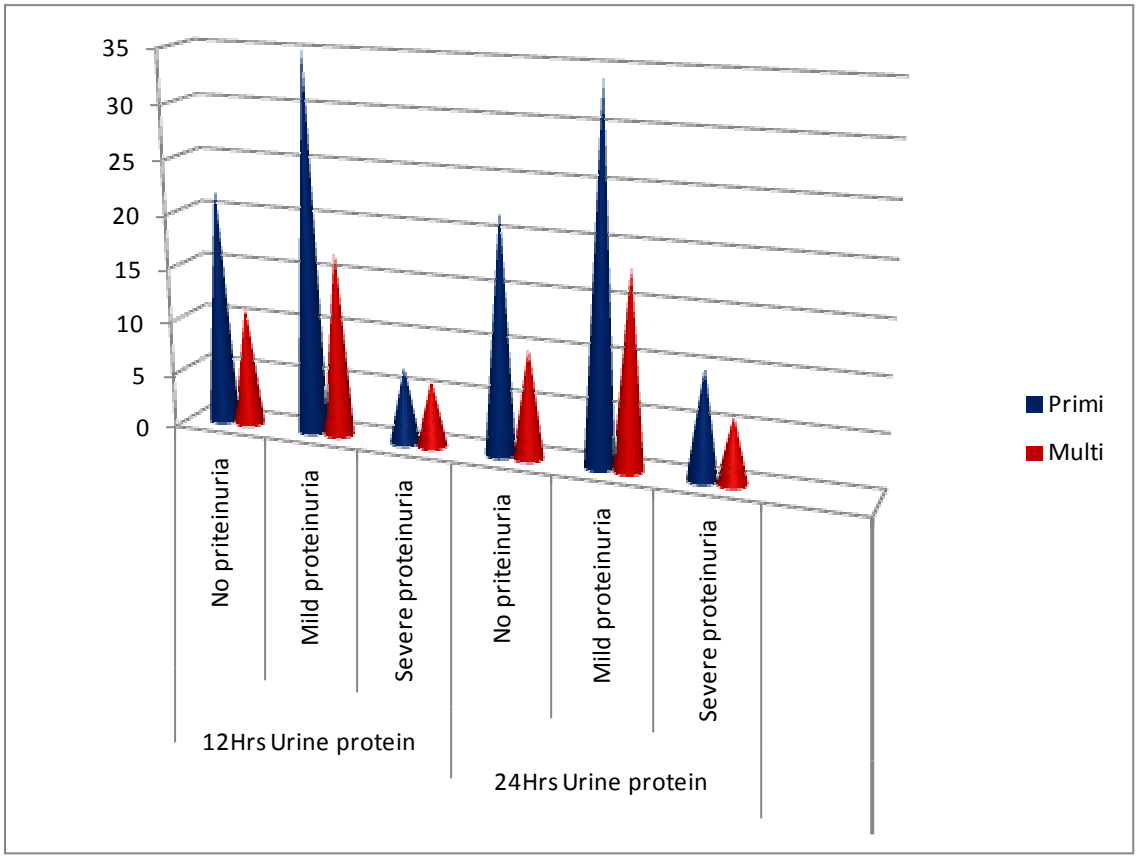


Table6: Distribution of cases according to urine Dipstick Analysis:

Urine Dipstick	No. of Patients
No Proteinuria	34
Mild proteinuria	61
Severe Proteinuria	5

According to urine dipstick trace is graded as no proteinuria, 1+, 2+, 3+ are graded as mild proteinuria, and 4+ is graded as severe proteinuria. In our study 34% of cases had no proteinuria, 61% had mild proteinuria and 5% had severe proteinuria.

Chart 5: Distribution of cases according to urine Dipstick Analysis:

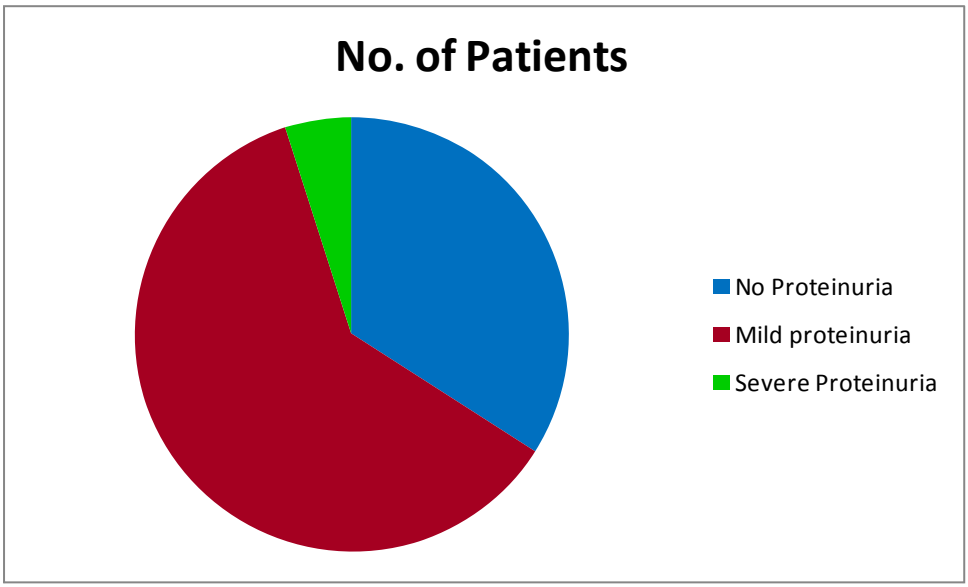


Table7: Correlation between Urine Dipstick with 12 and 24hrs Urine Protein:

	12Hrs Urine protein			24Hrs Urine Protein	
	No Proteinuria	Mild proteinuria	Severe Proteinuria	No Proteinuria	Mild proteinuria
No Proteinuria	32	2	0	32	2
Mild Proteinuria	2	48	11	0	50
Severe Proteinuria	0	1	4	0	0

In our study among the 34 patients who were diagnosed to have no proteinuria by dipstick had mild proteinuria in 2cases and no proteinuria in 32 cases in both 12 and 24hrs urine protein. Among 61 cases who were diagnosed to have mild proteinuria by dipstick 2 had no proteinuria, 48 had mild and 11 had severe proteinuria, whereas in 24hrs group 50 had mild and 11 had severe proteinuria. Out of 5 cases who were diagnosed to have severe proteinuria by dipstick 1 had mild and 4 had severe proteinuria by 12hrs, whereas in 24hrs group all 5 had severe proteinuria. There is a statistically significant (p= 0.001) difference between urine dipstick and 12/24hrs urine protein.

Chart 6: Correlation between Urine Dipstick with 12 and 24hrs Urine Protein:

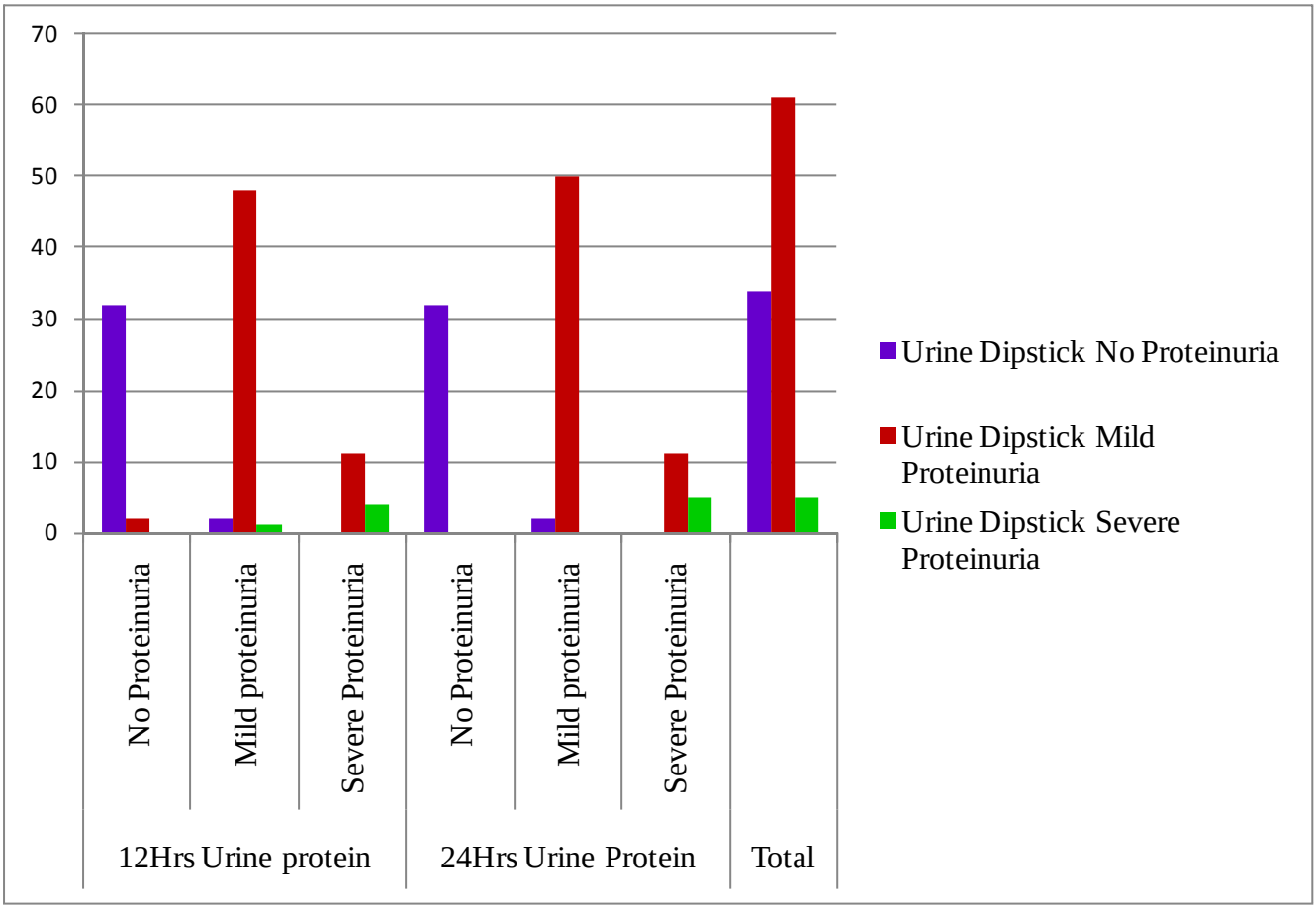


Table8: Distribution of cases according to the Gestational Age:

Gestational Age	No. Of Patients
<28 weeks	12
29-34 weeks	20
>34 weeks	68

Among the 100 cases studied 68 cases were > 34 weeks, 12 cases were <28 weeks and 20 cases were between gestational age 29 to 34 weeks. According to various studies hypertensive disorders of pregnancy are common during later periods of gestation which correlates with our study.

Chart 7: Distribution of cases according to the Gestational Age:

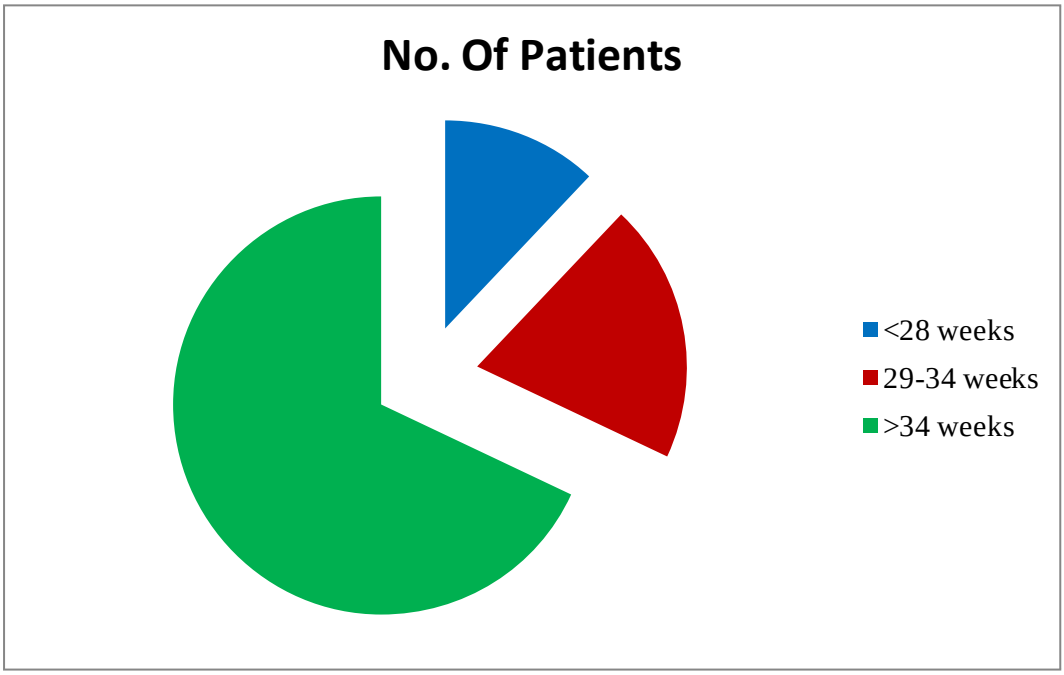


Table9: Distribution of proteinuria according to the Gestational Age:

	12Hrs Urine protein			24Hrs Urine Protein	
Gestational Age	No Protein uria	Mild protein uria	Severe Protein uria	No Protein uria	Mild protein uria
<28 weeks	2	4	6	2	4
29-34 weeks	8	9	3	7	10
>34 weeks	24	38	6	23	38
p value	0.007				

There is a good correlation between 12 and 24 hours urine protein in all degrees of proteinuria among various gestational age groups. Among the 24 cases in the > 34 weeks gestational age who had no proteinuria in 12 hrs sample 23 cases had no proteinuria even in the 24 hrs sample. Likewise mild and severe proteinuria also correlates in this gestational age.

Chart 8: Distribution of proteinuria according to the Gestational Age:

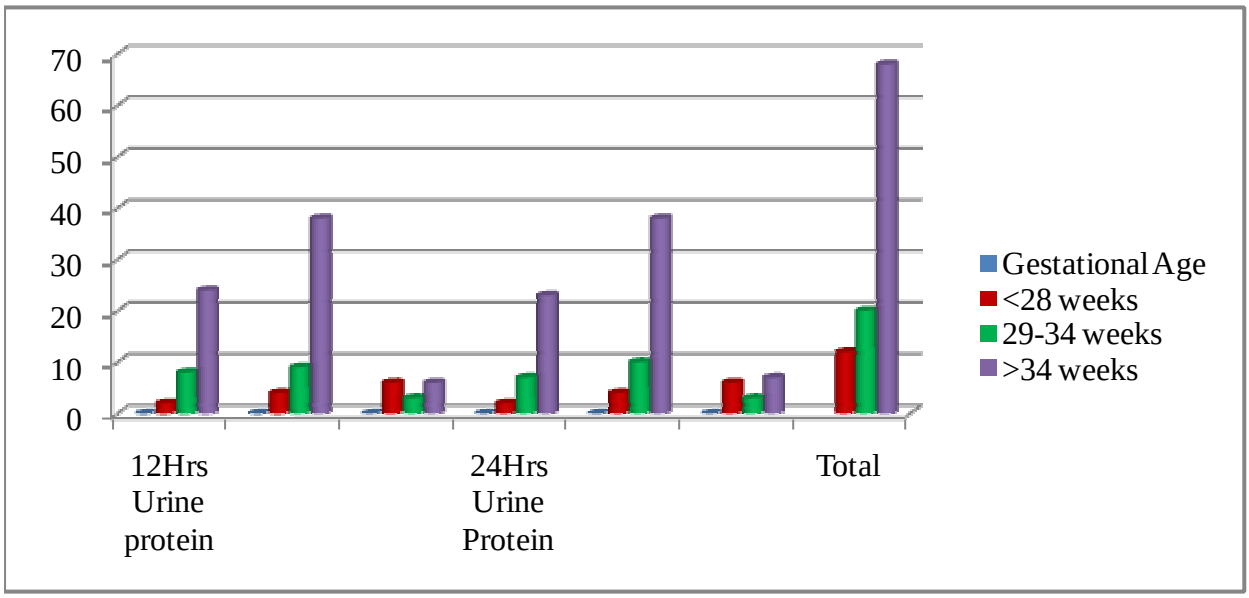


Table10: Distribution of cases according to the type of Hypertension:

Type of HT	No. of patients
GHT	34
PE	44
SPE	18
RPE	4

Among the 100 cases studied 34 had gestational hypertension, 44 had preeclampsia, 18 had severe preeclampsia and 4 had recurrent preeclampsia. The blood pressure among the cases ranges between 140/90 to 180/110 mmHg with mean systolic blood pressure of 149.10 mmHg and diastolic blood pressure of 94.10 mmHg.

Chart 9: Distribution of cases according to the type of Hypertension

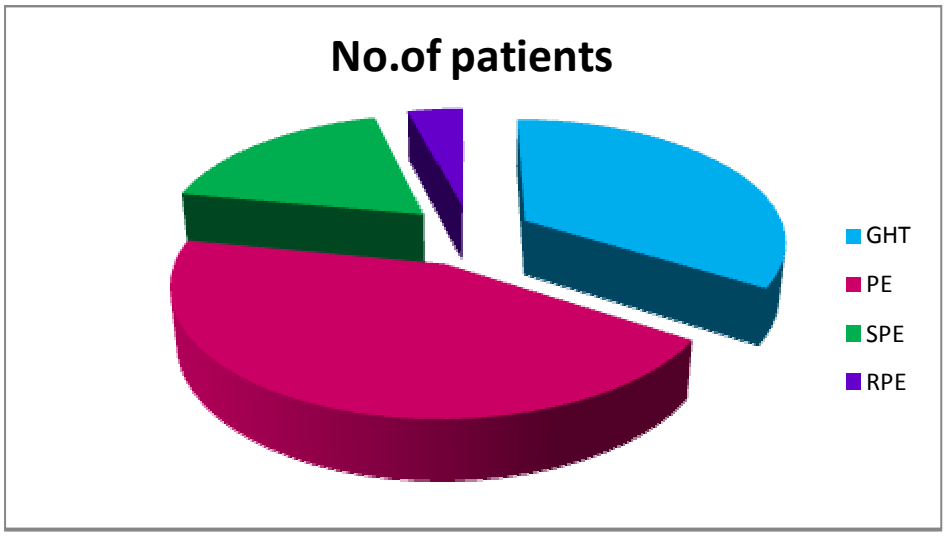


Table 11: Correlation between 12 and 24 hrs urine Protein and types of HT

Type Of HT	12 hrs Urine Protein			24 hrs Urine Protein		
	No Proteinuria	Mild proteinuria	Severe Proteinuria	No Proteinuria	Mild proteinuria	Severe Proteinuria
GHT	32	2	0	32	2	0
PE	2	42	0	0	44	0
SPE	0	3	15	0	2	16
RPE	0	4	0	0	4	0
			0.001			0.001

Out of the 34 patients who were diagnosed as gestational hypertension 32 had no proteinuria, 2 had mild proteinuria. Likewise 48 patients who were diagnosed as preeclampsia (PE&RPE) 2 had no proteinuria, 46 had mild proteinuria. Likewise 18 patients who were diagnosed as severe preeclampsia 3 had mild proteinuria, 15 had severe proteinuria. p value for both 12 and 24 hrs urine protein was 0.001, so there is statistically significant correlation between types of hypertension and 12/24 hrs urine protein.

Chart 10: Correlation between 12 and 24 hrs urine Protein and types of HT

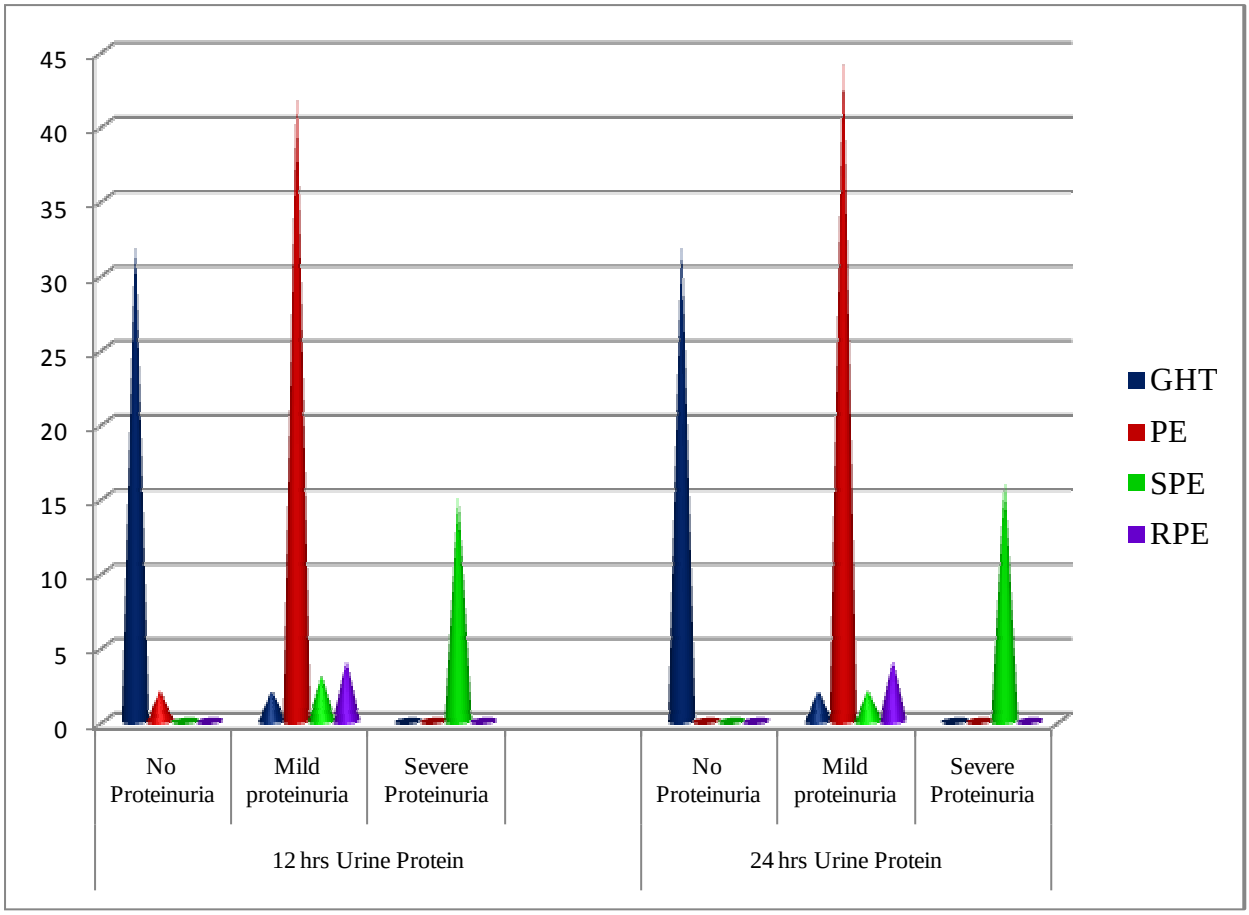


Table 12: Maternal Outcome among the cases:

Maternal Outcome	No.Of Patients
Good	84
Adverse	16

The overall maternal outcome was good in no proteinuria group in both 12 and 24 hrs urine protein study.The percentage of adverse maternal outcome was less in mild preeclampsia group when compared to severe preeclampsia in both 12

and 24 hrs steudy. The adverse maternal outcomes like terminatination of pregnancy on maternal or fetal grounds leading to increaser rate of caesarean section, starting Mgso4 regimen, antepartum eclampsia, abruption, HELLP syndrome.

Chart: 11: Maternal Outcome among the cases:

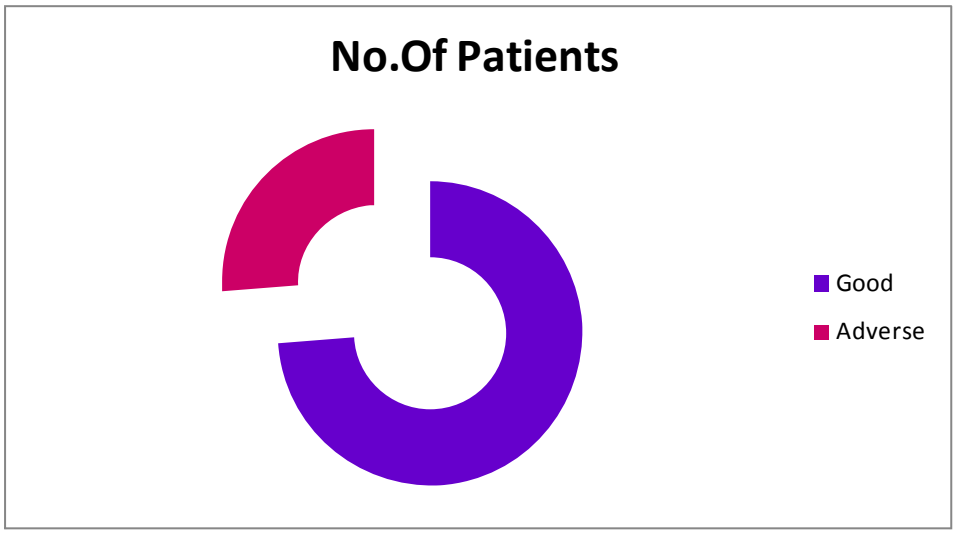


Table: 13: Correlation between Maternal outcome and severity of Proteinuria:

	12Hrs Urine protein			24Hrs Urine Protein	
	No Proteinuria	Mild proteinuria	Severe Proteinuria	No Proteinuria	Mild proteinuria
Good	34	45	5	32	47
Adverse	0	6	10	0	5

In the 12 hrs urine protein, out of the 34 patients with no proteinuria all the patients (100%) had good maternal outcome. Among the 51 patients with mild proteinuria only 6(11.7%) patients had adverse outcome. Whereas among 16 patients among severe proteinuria group 10 (66.7%) had adverse outcome. This is similar to the 24 hrs urine protein where among the 52 patients who had mild proteinuria 5(9 .6 %) had adverse outcome and among the 16 patients in severe preeclampsia group 11(68.7%) had adverse outcome.

Chart 12: Correlation between Maternal outcome and severity of Proteinuria

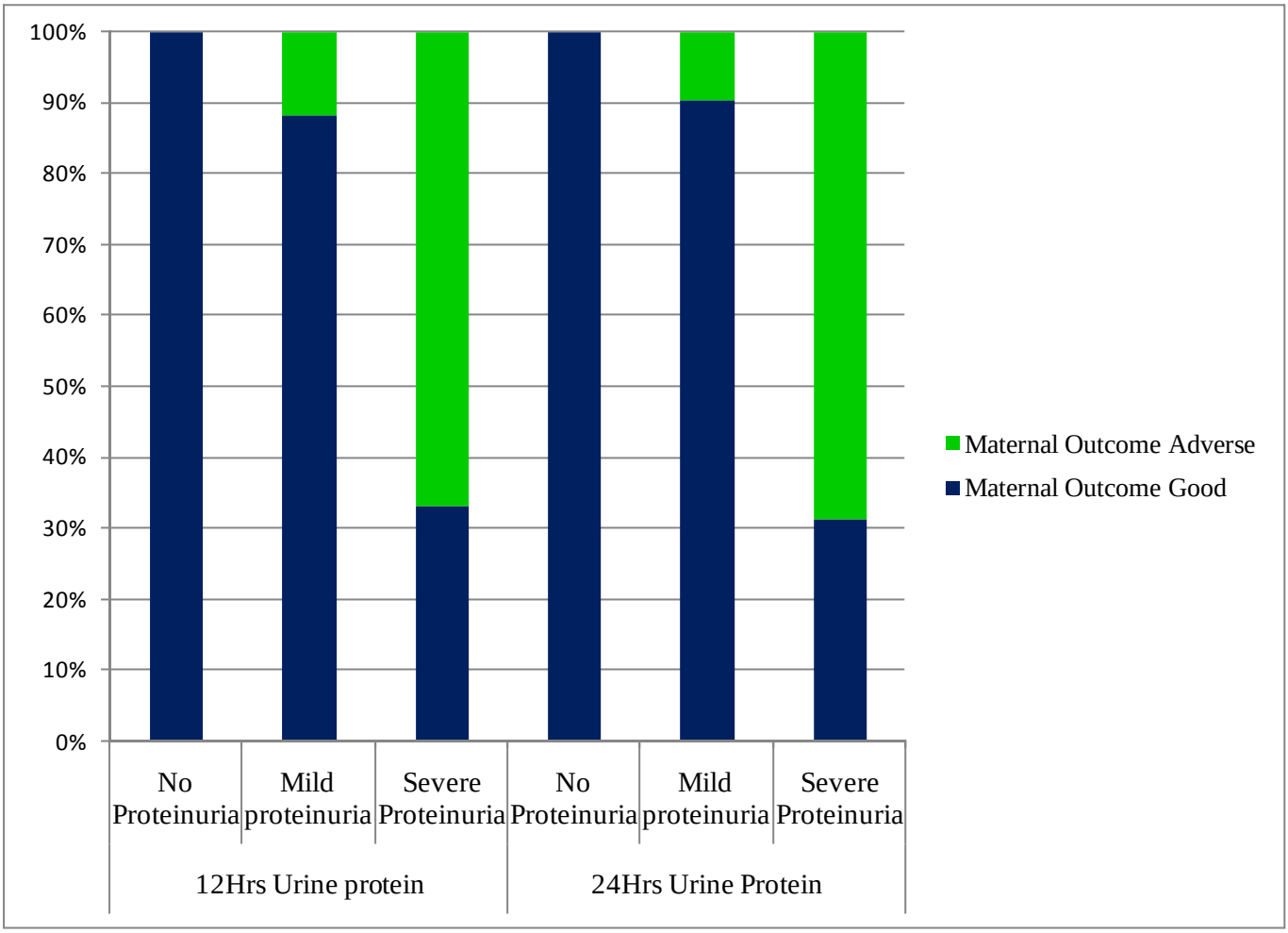


Table 14: Fetal outcome among Cases

Fetal outcome	No.Of Patients
Good	74
Adverse	26

The fetal outcome was good when there is no proteinuria, and fetal complications occur when there is mild or severe proteinuria.The adverse neonatal outcomes includes preterm delivery, low birth weight, IUGR , low APGAR score,NICU admission. Among the 100 patients neonatal outcome was affected in 26 cases.

Chart: 13: Fetal outcome among Cases:

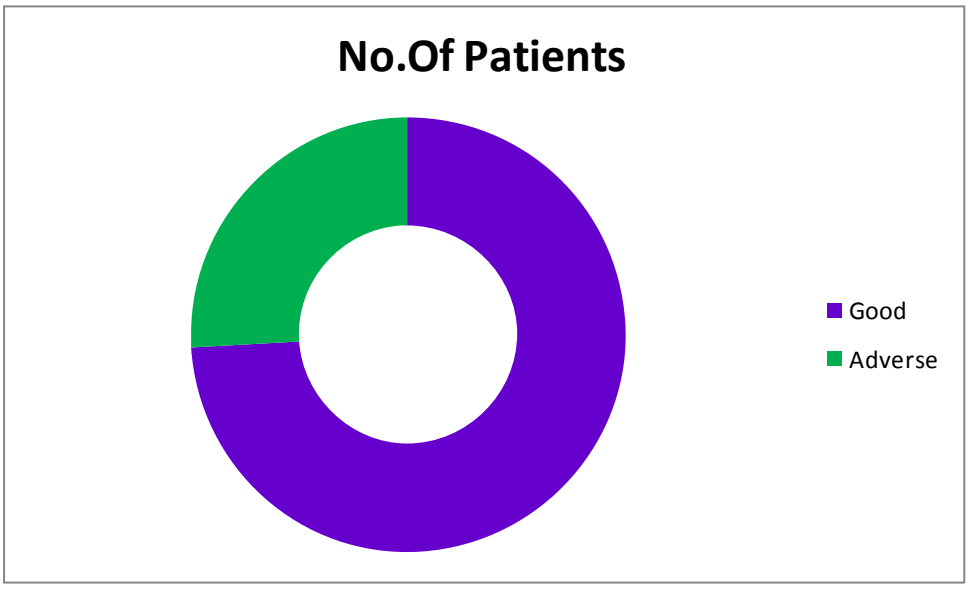


Table 15: Correlation between Fetal outcome and severity of Proteinuria

	12Hrs Urine protein		24Hrs Urine Protein		
	No Proteinuria	Mild proteinuria	No Proteinuria	Mild proteinuria	Severe Proteinuria
Good	34	39	32	41	1
Adverse	0	12	0	11	15

The above table shows that fetal outcome worsens with the degree of proteinuria the outcome is good when there is no proteinuria and most of the babies had an adverse outcome when there is severe proteinuria .In the 12 hrs sample in the severe proteinuria group 93.2% had adverse outcome which is very similar to to the 24 hrs urine sample which shows 93.7% adverse outcome in the severe preeclampsia group. In the mild proteinuria group adverse outcome occurs in 23.5% in the 12hrs study and 21.1% in the 24hrs study which is statistically significant with a P value of 0.001.

Chart 14: Correlation between Fetal outcome and severity of Proteinuria:

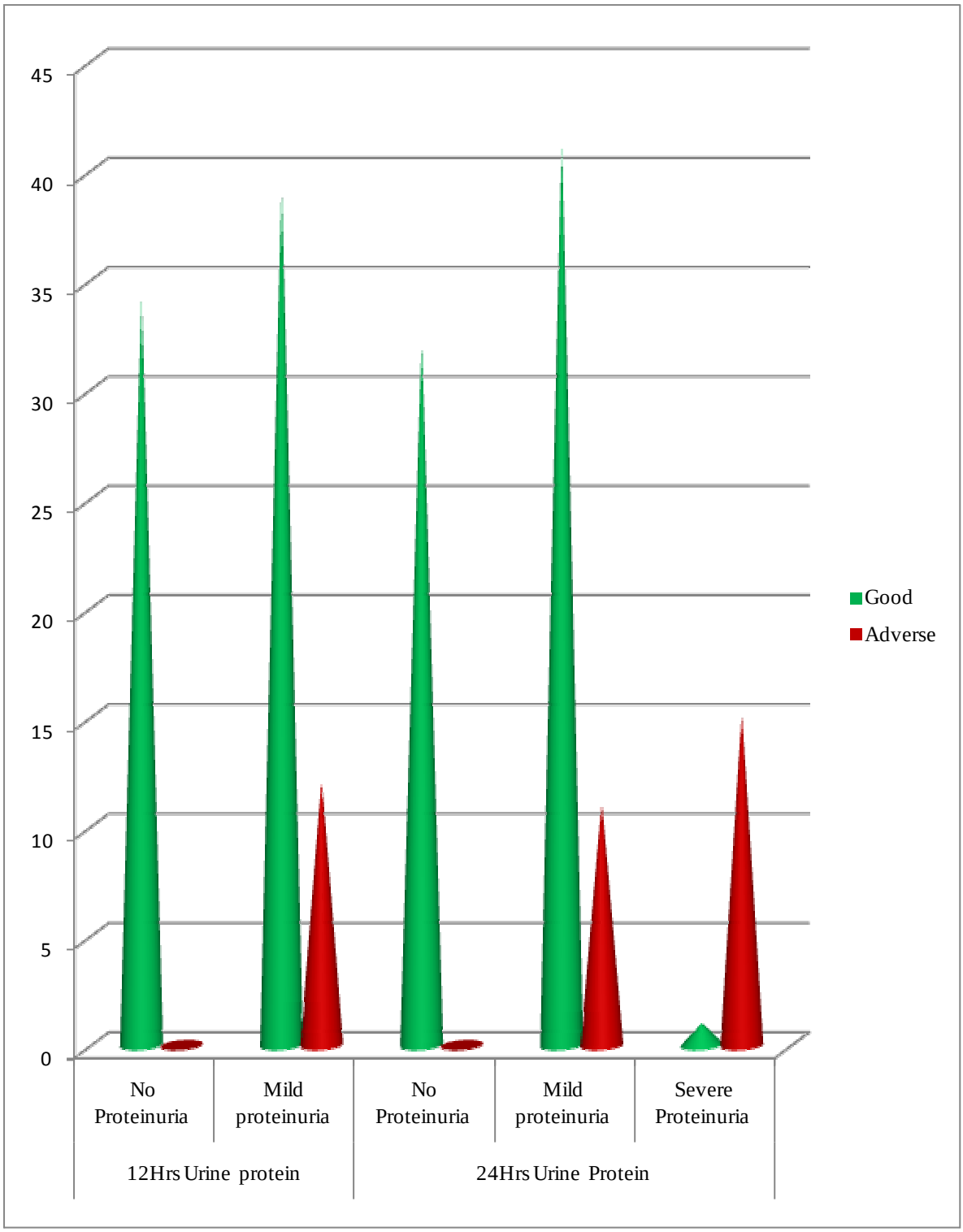


Table 16: Correlation between 12Hrs and 24Hrs urine Protein:

			24Hrs Urine Protein			Total
			No	Mild	Severe	
12Hrs Urine Protein	No	Count	32	2	0	34
		% within 12Hrs Urine Protein	94.1%	5.9%	.0%	100.0%
		% within 24Hrs Urine Protein	100.0%	3.8%	.0%	34.0%
	Mild	Count	0	49	2	51
		% within 12Hrs Urine Protein	.0%	96.1%	3.9%	100.0%
		% within 24Hrs Urine Protein	.0%	94.2%	12.5%	51.0%
	Severe	Count	0	1	14	15
		% within 12Hrs Urine Protein	.0%	6.7%	93.3%	100.0%
		% within 24Hrs Urine Protein	.0%	1.9%	87.5%	15.0%
Total		Count	32	52	16	100
		% within 12Hrs Urine Protein	32.0%	52.0%	16.0%	100.0%
		% within 24Hrs Urine Protein	100.0%	100.0 %	100.0%	100.0%

Table:17: Chi-Square Test for Comparing 12 and 24hours Urine Protein:

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	167.164(a)	4	.000
Likelihood Ratio	160.139	4	.000
Linear-by-Linear Association	88.586	1	.000

Among the patients who had no proteinuria in the 12 hrs study 94.1% had no proteinuria and 5.9% had mild proteinuria in the 24 hrs study.Among the

patients who had mild proteinuria in the 12 hrs study 96.1% had mildproteinuria and 3.9% had severe proteinuria in the 24 hrs study.Among the patients who had severe proteinuria in the 12 hrs study 6.7% had mildproteinuria and 93.3% had severe proteinuria in the 24 hrs study.The study shows a pearson chi-square value of 0.001 which is statistically significant.Thus 12 and 24 hrs sample coincides in all degrees of proteinuria in all ages at all gestational ages and various degrees of hypertension.

Table:18: PEARSONS Correlation coefficient for Comparing 12 and 24hours Urine :

		Value	Asymp. Std. Error(a)	Approx. T(b)	Approx. Sig.
Interval by Interval	Pearson's R	.946	.024	28.873	.000(c)
Ordinal by Ordinal	Spearman Correlation	.950	.022	30.245	.000(c)
N of Valid Cases		100			

The pearson's correlation coefficient (r) value comparing 12 and 24 hrs urine protein was 0.946 which correlates with other studies.

STUDIES	**CORRELATION COEFFICIENT (r)**
SOGRE RABIE	**0.86**
CHRISTINA TUN	**0.99**
RANGASAMY SAVITHA	**O.96**
OUR STUDY	**0.94**

Chart 15: Correlation between 12Hrs and 24Hrs urine Protein:

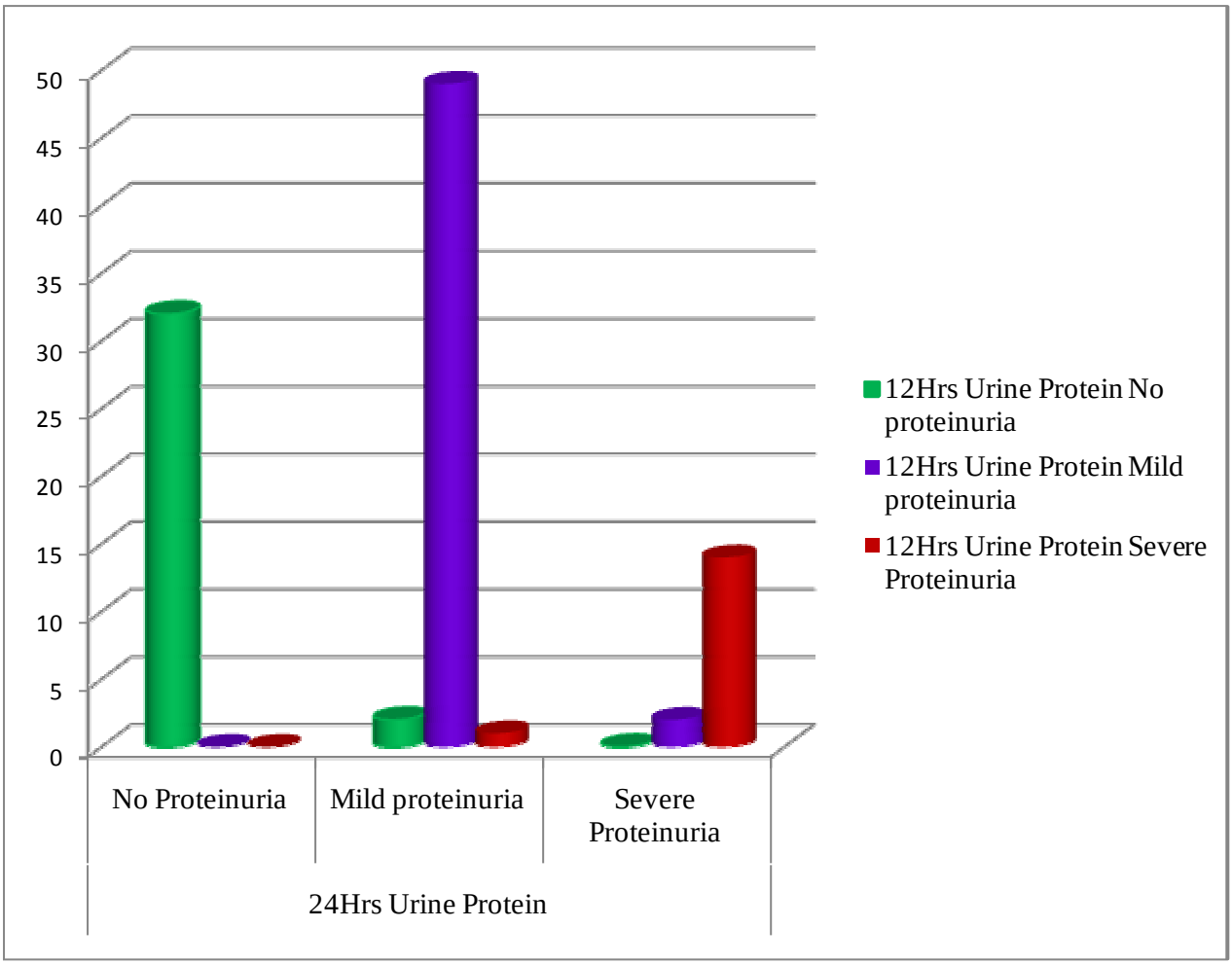


Table 18: Statistical Significance

			24Hrs Urine Protein		Total
			Normal	Abnormal	
12Hrs Urine Protein	Normal	Count	32	2	34
		% within 12Hrs Urine Protein	94.1%	5.9%	100.0%
		% within 24Hrs Urine Protein	98.1%	2.9%	34.0%
	Abnormal	Count	0	66	66
		% within 12Hrs Urine Protein	.0%	99.0%	100.0%
		% within 24Hrs Urine Protein	.0%	97.1%	66.0%
Total		Count	32	68	100
		% within 12Hrs Urine Protein	32.0%	68.0%	100.0%
		% within 24Hrs Urine Protein	100.0%	100.0%	100.0%

The above table explains the statistical significance of the study comparing the accuracy of 12 hrs urine protein with 24 hrs urine protein in predicting proteinuria with a Sensitivity of 98.1% , Specificity of 97.1%, Positive predictive value of 94.1%, Negative predictive value 99%.

VI. DISCUSSION

In our study 100 patients with hypertensive disorders of pregnancy were enrolled after fulfilling the inclusion & exclusion criteria.

INCIDENCE:

Study	Incidence of Preeclampsia
Mudaliar & Menon	10%
Sanchez Ramos	14%
Ritu Kamare et al	13.6%
Rodriguerz et al	10.6%
Present Study	9.6%

In our hospital the incidence of preeclampsia was 9.6%.

AGE INCIDENCE:

Women of different age group were included in the study. Out of the 100 patient 67% of them were in the age group between 17 to 21 &>28 years which is in accordance with the study of Mac Gillivary (1958) which showed a J shaped curve between the relationship of maternal age & incidence of hypertensive disorders of pregnancy.

Similarly a study by Remich (1989) in Virginia showed a significant relationship between maternal age & incidence of pre eclampsia.

Hansen reported 2 to 3 – fold increase in preeclampsia over 30 years.

PARITY:

Hypertensive disorders of pregnancy were common in primi. Sibai& colleagues (1993) confirmed the higher risk of developing hypertension in the first pregnancy. According to a study conducted in cape peninsula (1979-81) there was double the incidence of hypertension & proteinuria in primi.

In our study primi constitutes 66% with a p value of <0.001which is statistically significant, it correlates with the studies.

SOCIO ECONOMIC STATUS:

Low socio economic status is reported to have high incidence of hypertensive disorders of pregnancy. This may be attributed to their poor nutrition.

In our study most of them belong to class IV & V socio economic atatus.Dr.Dawn (1964) & Dr.Richares nage (1981) showed that maternal nutririon exerts its regulating effect on the fetal growth through its effect on blood volume, blood pressure & utroplacental blood flow. This correlates with our study.

GESTATIONAL AGE:

The averagegestational age in our study is 35.1 weeks. About 68% cases develop HT >34 weeks, According to various studies hypertensive disorders are common during later periods of gestation.

Correlation between dipstick analysis & 12/24 hours urine Protein:

In our study there is poor correlation between urine dipstick & 12/24 hours urine protein with ap value of 0.001 which is statistically significant.

Suden et al showed that the high number of false negatives with urine dipstick when compared to 24 hours urine measurement.

Mayer et al found that dipstick is apoor predictor of absent or severe proteinuria.

Jasone et al showed dipstick values of trace or 1+ correlates poorly whereas at higher degrees it correlates better.

According to the type of hypertension:

Among the 100 patients in the study, 34 had gestational hypertension, 48 had preeclampsia of which 4 patients had recurrent preeclampsia & 18 had severe preeclampsia. In all types of hypertensive disorders of pregnancy there is a very good correlation between 12&24 hours urine protein with a p value of 0.001, which is statistically significant.

Maternal Outcome:

The maternal complications that are common in preeclampsia & proteinuria are eclampsia, abruption placenta mgso4 regimen, DIC, HELLP syndrome. There is increase in rate of termination of pregnancy for maternal interest by induction of labour which ends in increase in rate of caesarean section and preterm delivery.

Eclampsia:

Hibaral (1973) showed that about 20% of maternal death in preeclampsia is due to eclampsia. In a study conducted by Basker Rao in 1971 incidence of eclampsia in hospitalised patients was 3% & 6% on unbooked case. In our study eclampsia occurs in 4 cases i.e., 4% which correlates with the study. All these cases belong to severe proteinuria group.

Abruptio Placenta:

In 1997 a study conducted by Hollier and Cunningham showed that the risk of abruption was 2.1 to 4 %. In our study incidence of abruption was 2%

Which correlates with that study.

Mgso4 Regimen:

It is an anticonvulsant used in the treatment of severe preeclampsia out of the 100 patients in our study mgso4 regimen was started in 8 cases. All of these belong to severe proteinuria group.

Course of Labour:

In 1966 shiring showed that preeclampsia is a common cause of induction of labour. In our study, pregnancy was terminated for maternal and fetal intrest in 17 cases which leads to increased rate of caesarean section and preterm birth

Fetal Outcome:

The common complications associated with severe proteinuria are low birth weight, IUGR, oligohydramnios, preterm birth, low APGAR scores. The incidence of IUGR in our study was 8% , 15% preterm birth, 2% still born, 6% oligohydramnios. In a study conducted by Doss et al (1998) prematurity was 17.6% which correlates with our study. According to mudaliar and menon the incidence was 22%.

Indir (1984) showed that incidence of IUGR in severe proteinuria is 8% which correlates with our study.

Correlation between 12 hrs and 24 hrs urine protein:

There is a significant correlation between 12 and 24 hrs urine protein in all types of hypertension (GHT, PE, SPE) and in all degrees of proteinuria (No, mild , severe). The study shows a Pearsons Chi square value of 0.001 which is statistically significant.

Sogre Rabie	P < 0.01
Otero Pinto	P<0.01
Christina Tun	P <0.001
Rangasamy Savitha	P <0.0001
Irrvae	P <0.02
Our Study	P <0.001

SENSITIVITY AND SPECIFICITY :

The datas thus collected are analysed using appropriate statistical methods and sensitivity and specificity are tabulated.

STUDY	SENSITIVITY %	SPECIFICITY %	POSITIVE PREDICTIVE VALUE %	NEGATIVE PREDICTIVE VALUE %
Minoo Rajaei (n=63)	83	91	80	82
Sogre Rabiae (n=57)	94	99	98	99
Christina Tun (n=102)	96	100	100	98
Sogre Fillahi (n= 65)	82	88	85	86
Irruae (n=65)	96	98	98	96
Our Study (n=100)	98	97	94	99

CORRELATION COEFFICIENT:

The correlation coefficient r value comparing 12 and 24 hrs urine protein was 0.94 which is similar to other studies.

STUDIES	**CORRELATION COEFFICIENT (r)**
SOGRE RABIE	**0.86**
CHRISTINA TUN	**0.99**
RANGASAMY SAVITHA	**0.96**
OUR STUDY	**0.94**

SUMMARY

***** The study is conducted in hospitalised patients above 20 weeks of gestation with hypertensive disorders of pregnancy.

* Since the level of urinary protein excretion has considerable clinical implications in the course of pregnancy and on the perinatal and neonatal outcome the early detection of even minor degrees of proteinuria is important.

* For years 24 hrs urine collection has been the gold standard for quantitation of proteinuria in the management of women with preeclampsia, however this method is cumbersome subjective to collection errors requires good patient compliance and results in the delay in the diagnosis > 24 hrs from the start of collection.

* Proteinuria is proved to be an important predictor of maternal and fetal outcome. As the degree of proteinuria increases there is increase in neonatal and maternal morbidity and mortality.

***** This study shows that there is high degree of correlation between 12 and 24 hrs urine protein in all types of hypertension and all degrees of proteinuria.

* Our studyshowed that 12 hr sample correlated with 24 hrs sample for patients with no proteinuria ($p < 0.001$) mild proteinuria ($p<0.001$), severe proteinuria ($p < 0.001$) which is statistically significant.

* The correlation coefficient (r) between 12 and 24 hrs urine protein is 0.94 which is statistically significant.

* The statistical significance of the study comparing the accuracy of 12 hrs urine protein with 24 hrs urine protein in predicting proteinuria with a Sensitivity of 98.1% , Specificity of 97.1%, Positive predictive value of 94.1%, Negative predictive value 99%.

VII. CONCLUSION

Our study showed that `there is a high correlation of 12 hrs urine protein >165mg with a 24 hrs urine protein>300mg in all degrees of proteinuria and all types of hypertension.

The present study indicates that this method of quantification of proteinuria when properly interpreted can provide valuable information that for clinical purposes is a satisfactory substitute for the determination of protein excretion in a 24 hr collection.

VIII.ANNEXURE

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B.PROFORMA SHEET

NAME:

AGE: IP NO: UNIT: S.NO:

DATE OF ADMISSION:

DATE OF DELIVERY:

DATE OF DISCHARGE:

SOCIOECONOMIC STATUS:

LAST MENSTRUAL PERIOD:

EXPECTED DATE OF DELIVERY:

OBSTETRIC CODE:

G P L A

ADMITTED FOR: Safe Confinement

Labour pains/ draining/bleeding/pedal edema

Headache/blurring of vision/ oliguria

Convulsions

MENSTRUAL HISTORY: Regular/ Irregular

LMP:

MARITAL HISTORY: Married since

Consanguinity-

OBSTETRIC HISTORY: G P L A

Nature of previous deliveries:

Labour natural/ caesarean section /Termination

PRESENT PREGNANCY:

I TRIMESTER: Fever, Drug intake, Radiation, Bleeding per vaginum.

II TRIMESTER: Bleeding, Pedal Edema, Vomiting, Headache, Visual Disturbances.

III TRIMESTER: Bleeding, Pedal Edema, Vomiting, Headache, Visual Disturbances.

PAST HISTORY:

Gestational Hypertension, Preeclampsia, Renal Disease

FAMILY HISTORY:

Hypertension, epilepsy, Diabetes

MEDICAL HISTORY:

Chronic Hypertension, Diabetes Mellitus, Renal Disease

GENERAL EXAMINATION

Built:

Pallor:

Jaundice:

Pedal Edema:

VITALS:

Pulse Rate:

Blood Pressure:

I$^{st\ Reading}$:

II[nd] Reading:

CARDIOVASCULAR SYSTEM:

RESPIRATORY SYSTEM:

PER ABDOMEN:

Height of uterus:

Acting/ not acting:

Tense /Tender:

Liquor:

INVESTIGATIONS:

Urine: Alb:

Sugar and deposits:

Blood: Hb %:

Total count:

Platelets:

Gr/ Typing:

Blood Urea:

Blood Sugar:

Serum Creatinine:

Liver function Tests:

Serum Bilirubin:

Serum Proteins:

SGOT:

SGPT:

SAP:

Serum uric acid :

Serum fibrinogen:

12hrs urine protein:

24hrs urine protein:

Fundus

TREATMENT:

Antihypertensive:

Magnesium sulphate Regimen:

LABOUR:

Mode of Delivery:

Labour Natural/ Caesarean Section/ Termination

COMPLICATIONS:

MATERNAL:

Eclampsia

Abruption

HELLP

Pulmonary Edema

BABY:

Alive/ Stillborn/ intra uterine Death

C.ABBREVATIONS

SBP –Systolic Blood Pressure

DBP –Diastolic Blood Pressure

GHT – Gestational Hypertension

PE –Preeclampsia

PCR – Protein Creatinine Ratio

IUGR –Intra Uterine Growth Restriction

TXA2 – Thromboxane A2

PGI2 –Prostaglandin I2

Sflt –Soluble fms like Tyrosine Kinase 1

S(Eng)- Soluble Endoglin

AST –Aspartate transaminase

ALT –Alanine Transaminase

LBW – Low Birth Weight

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Comparing the accuracy of 12hrs urine protein with 24 hrs urine protein in predicting proteinuria in hypertensive disorders complicating pregnancy

Principal Investigator : Dr.P. Nirmala Devi

Designation : PG in MS(OG)

Department : Department of OG
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.04.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

12/8/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MASTER CHART FOR 100 CASES OF HYPERTENSIVE DISSORDERS OF PREGNANCY

Sl.No	Name	Age	IP No	Obstetric Code	Gestational Age	Urine Albumin	SBP	DBP	Type of HT	Platelet Count	Uric Acid	12Hrs Urine Protein	24Hrs Urine Protein	Mode of delivery	Maternal outcome	Fetal outcome
1	Vasanthi	28	4708	Primi	36	1+	140	100	PE	2.5	5.2	232	450	LN	Good	Good
2	Jayashree	25	4846	Primi	26	4+	160	100	SPE	1.7	7.2	1600	3500	LSCS	Abruption	Preterm
3	Allice Mary	30	4088	G2P1L1	Term	1+	150	90	RPE	2	5.3	260	525	LSCS	Good	Good
4	Karuna	28	4833	Primi	32	3+	160	90	SPE	2.3	7	1700	3200	LN	Good	Preterm
5	Jothi	26	4922	G2P1L1	Term	1+	150	90	RPE	2.1	6.5	378	785	LN	Good	Good
6	Helen	30	4955	G2P1L1	36	Trace	140	90	GHT	2.8	4	78	165	LN	Good	Good
7	Kushboo	19	4558	Primi	Term	1+	160	100	PE	3	4.5	190	364	LN	Good	Good
8	Nathiya	24	4584	G2P1L1	36	2+	160	100	PE	2.9	4.8	218	420	LSCS	Good	LBW
9	Murugavalli	21	4828	Primi	Term	2+	160	100	PE	2.4	7.2	300	608	LSCS	MGSO4 regimen	IUGR
10	Vijayamma	20	4969	Primi	Term	1+	140	90	PE	2.5	4.5	770	1500	LN	Good	Good
11	Indumathy	27	5027	G2P1L1	Term	Trace	140	90	GHT	2.8	3.8	40	88	LN	Good	Good
12	Ramalakshmi	25	5164	G2P1L1	34	1+	140	90	PE	2.4	5.6	204	410	Conservative management		
13	Jagadeshwari	20	5004	Primi	24	3+	180	110	SPE	2	8.2	1948	3950	Termination		Stillborn
14	Bavani	19	5009	Primi	36	1+	150	100	PE	3	4.5	250	508	LN	Good	Good
15	Shobana	26	5669	Primi	32	3+	180	100	SPE	2.3	7.8	1850	3800	Termination	MGSO4 regimen	Preterm
16	Shanavas	28	5736	Primi	34	1+	150	100	PE	2.1	6.8	296	606	LSCS	Good	Preterm
17	Gowri	21	5743	Primi	34	1+	150	90	PE	2.5	4.2	185	350	Conservative management		
18	Revathy	19	5755	Primi	36	1+	150	90	PE	1.8	5	208	420	LN	Good	Good
19	Sumathy	27	6079	G2P2L2	Term	Trace	140	90	GHT	2.8	3.6	142	175	LN	Good	Good
20	Fathima	28	6218	Primi	36	1+	140	90	PE	2.3	4.1	255	520	LN	Good	Good
21	Lakshmi	30	6416	G2P1L1	34	Trace	130	90	GHT	3	5.1	45	92	EM		
22	Amutha	25	6287	Primi	Term	2+	150	90	PE	2	4	275	575	LN	Good	Good
23	Gurupriya	21	6485	Primi	Term	1+	160	90	PE	2.5	4.2	416	848	LN	Good	Good
24	Badurunisha	21	6715	Primi	36	Trace	140	90	GHT	3.2	4.8	105	220	LN	Good	Good

25	Menaka	32	7224	Primi	32	Trace	130	90	GHT	1.9	4.5	42	90	EM		
26	Girija	26	7392	G2P1L1	28	3+	160	100	SPE	2.1	7.2	1705	3450	LSCS	IE	Preterm
27	Amulu	29	7758	G2P1L1	32	Trace	140	90	GHT	2.5	3.8	74	156	EM		
28	Karpagam	24	7985	Primi	36	Trace	140	90	GHT	3	5	110	225	LN	Good	Good
29	Kasthuri	40	7325	G2P1L1	32	Trace	140	90	GHT	2.8	4.2	110	230	EM	Good	
30	Yasmine Banu	22	8044	Primi	32	Trace	140	90	GHT	3.2	4.8	168	320	EM		
31	vimala	19	8286	Primi	Term	Trace	140	90	GHT	3	3.6	180	338	LN	Good	Good
32	Bavani	20	8266	Primi	28	Trace	150	90	GHT	2.6	5.2	135	254	EM		
33	Syed sulthana	20	8358	Primi	32	Trace	140	90	GHT	2.8	5.2	115	206	EM		
34	Meena	20	9050	Primi	36	1+	150	90	PE	2.4	6	225	442	LSCS	Good	Preterm
35	Shamim fathima	27	9091	G2P1L1	34	2+	160	100	PE	2	7.9	456	940	LSCS	Good	LBW
36	Lakshmi Devi	24	9048	G2P1L1	36	1+	140	100	PE	2.2	6.5	610	1200	LN	Good	Preterm
37	Chinthamani	21	9142	Primi	Term	Trace	140	90	GHT	2.9	5.2	116	240	LN	Good	Good
38	Saritha	24	9512	G2P1L1	Term	2+	140	90	PE	3	6	418	848	LN	MGSO4 regimen	Good
39	Jayachithra	21	9644	Primi	Term	Trace	150	90	GHT	2.9	5.6	104	206	LN	Good	Good
40	Abhitha	27	9649	G3P1L1A1	36	1+	150	90	PE	3.1	6.1	198	390	LSCS	Good	Good
41	Rajeswari	33	9648	G3P2L2	28	3+	160	100	SPE	2	8	1900	3600	Termination	MGSO4 regimen	IUGR
42	Valli	20	9878	Primi	28	Trace	140	90	GHT	1.7	4	76	146	EM		
43	Malathi	25	9906	Primi	Term	3+	160	110	SPE	1.9	7.5	2198	4250	Termination	MGSO4 regimen	IUGR
44	Saritha	19	9940	Primi	32	Trace	140	90	GHT	2	5.2	110	208	EM		
45	Priya	21	9942	Primi	Term	4+	160	100	SPE	2.9	7.2	2236	4450	Termination	Good	IUGR
46	Rekha	27	9988	Primi	36	Trace	140	90	GHT	3.4	5	110	206	LN	Good	Good
47	Lakshmi devi	24	10128	G2P1L1	34	1+	160	100	RPE	3	6.1	230	444	Termination	Good	Preterm
48	Hemavathi	30	10204	Primi	28	4+	150	100	SPE	2	7.6	1990	4010	Termination	IE	Stillborn
49	Jaylakshmi	21	10215	Primi	Term	1+	140	100	PE	2.7	5.2	266	546	LN	Good	Good
50	Suriya banu	25	10836	G3P1L1A1	Term	2+	160	100	SPE	1.9	6.8	1582	3146	LSCS	Abruption	IUGR
51	MUthammal	26	10878	G2P1L0	36	2+	160	100	SPE	3	7.2	1730	2400	Termination	IE	Preterm
52	Dhanalakshmi	28	10914	Primi	Term	2+	160	100	PE	2.5	7.6	462	880	LN	Good	Good

53	Aarthi priya	25	10940	Primi	Term	1+	160	110	SPE	2.7	6.9	1380	2600	LN	MGSO4 regimen	Good
54	Mehaboo bee	20	11014	G2P1L1	Term	1+	160	100	RPE	3	6	930	1755	LSCS	Good	Good
55	Vijayalakshmi	19	11213	G2P1L1	Term	Trace	140	90	GHT	2.6	5	122	226	LN	Good	Good
56	Ilavarasi	27	12746	G2P1L1	Term	1+	160	90	PE	1.8	4.8	238	456	LN	Good	Good
57	Kudiya devi	20	12830	G2A1	36	3+	160	110	SPE	2	6.1	1700	3360	Termination	Oligohydramnios	Preterm
58	Mathubala	19	13010	Primi	36	4+	160	100	SPE	2.3	6.5	2246	4200	Termination	MGSO4 regimen	IUGR
59	Rajeswari	21	13022	G2P1L1	Term	Trace	140	90	GHT	3	4.8	122	228	LN	Good	Good
60	Devi	26	13077	G2P1L1	Term	Trace	140	90	GHT	3.2	4.8	130	244	LN	Good	Good
61	Devika	25	13098	G2P1L1	32	2+	150	100	PE	2	5.2	260	505	Termination	IE	Preterm
62	Devi	31	13167	Primi	36	1+	150	90	PE	2.9	5	400	744	LN	Good	Good
63	Shahana Begam	20	13189	Primi	36	3+	160	100	SPE	2.6	7	1730	3200	Termination	MGSO4 regimen	Preterm
64	Devi	26	13210	G2P1L1	28	4+	160	100	SPE	2.5	7.1	1980	3740	Termination	Good	IUGR
65	Usha	20	13244	Primi	Term	Trace	150	100	GHT	1.95	5	114	220	LN	Good	Good
66	Nithya	19	13258	Primi	Term	Trace	140	90	GHT	2	4.5	150	280	LN	Good	Good
67	Yasmin Begum	21	13185	G2P1L1	Term	1+	150	90	PE	2.6	6	265	508	LSCS	Good	Good
68	Kanchana	26	13385	Primi	Term	Trace	140	90	GHT	3.4	4.8	92	176	LN	Good	Good
69	Vijayakumari	28	13447	Primi	36	1+	140	90	PE	2.5	4	350	668	Termination	Good	Good
70	Sathya	20	13448	Primi	Term	2+	150	100	SPE	2	6.1	1850	3400	LSCS	Oligohydramnios	Good
71	Latha	33	13559	Primi	36	1+	160	100	PE	2.1	5.6	920	1746	Termination	Good	Preterm
72	Seetha	21	13578	Primi	Term	1+	150	90	PE	2.5	5	675	1256	LN	Good	Good
73	Ramya	19	13638	Primi	Term	Trace	140	90	GHT	2.3	4.8	52	102	LN	Good	Good
74	Vennila	24	13756	G4P2L1A1	Term	1+	150	90	PE	1.9	4	400	778	LN	Good	Good
75	Sudha	27	13757	G3P2L2	34	3+	150	110	SPE	1.8	6	2280	4200	Termination	Good	Preterm
76	Pallavi	20	13714	Primi	Term	Trace	140	90	GHT	2.2	4.6	135	256	LN	Good	Good
77	Gajalakshmi	30	13767	G2P1L1	36	1+	160	100	PE	2.6	5.2	345	660	Termination	Good	Preterm
78	Kavitha	20	13786	Primi	Term	Trace	140	90	GHT	2.9	4	150	286	LN	Good	Good
79	Meenakshi	28	13941	Primi	Term	1+	160	100	PE	2	5	698	1300	LN	Good	Good
80	Vasanthi	20	14337	Primi	Term	Trace	140	90	GHT	2.6	4.4	100	185	LN	Good	Good

81	Thamena	21	14351	Primi	Term	Trace	150	90	GHT	2.3	4.8	80	150	LN	Good	Good
82	Karthika	20	14364	G2P1L1	Term	Trace	150	90	GHT	2.1	4.2	120	225	LN	Good	Good
83	Vijayalakshmi	20	14366	Primi	Term	1+	140	90	PE	2	5.2	200	380	LN	Good	Good
84	Jayalakshmi	21	14368	Primi	32	Trace	140	90	GHT	3.1	4	110	200	EM		
85	Priyanka	20	14367	Primi	Term	Trace	140	90	GHT	3.4	4.6	146	280	LN	Good	Good
86	Nandhini	17	14383	Primi	Term	Trace	140	90	GHT	2.4	5	150	186	LN	Good	Good
87	Revathy	25	14423	Primi	36	Trace	140	90	GHT	2.8	4.3	104	198	LN	Good	Good
88	Nalini	20	14465	Primi	26	1+	150	90	PE	2.6	4.2	210	408	EM		
89	Vidhya	21	14482	Primi	32	1+	150	90	PE	3	4.8	294	560	EM		
90	Rani	26	14498	G2P1L1	34	1+	150	100	PE	2.7	4.5	156	308	EM		
91	Bavani	30	14513	Primi	26	1+	150	90	PE	3.2	3.8	166	326	EM		
92	Prema	20	14525	Primi	Term	1+	160	90	PE	3.7	5.2	428	790	LN	Good	Good
93	Anu	24	14587	Primi	32	1+	150	90	PE	2	5.6	200	380	EM		
94	Barathi	25	14599	Primi	36	2+	160	90	PE	3.1	5.3	412	805	LN	Good	Good
95	Ramya	19	14619	Primi	Term	2+	160	90	PE	2.9	4.8	780	1500	LN	Good	Good
96	Vinodini	18	14676	Primi	24	1+	140	90	PE	2.8	3.9	160	310	EM		
97	Anjali	26	14688	Primi	36	1+	140	90	PE	2.6	4.1	256	480	LN	Good	Good
98	Angammal	27	14690	Primi	Term	2+	150	90	PE	2.8	4.6	370	690	LN	Good	Good
99	Uma	24	14709	G2P1L1	Term	2+	150	100	PE	3.2	5.2	420	786	LN	Good	Good
100	Vanitha	25	14756	Primi	28	1+	140	90	PE	3.4	5.1	175	320	EM		

GHT - Gestational Hypertension, PE – Preeclampsia, SPE – Severe Preeclampsia, RPE – Recurrent Preeclampsia, SBP – Systolic Blood Pressure, DBP – Diastolic Blood Pressure, IUGR – Intra Uterine Growth Restriction , EM – Expectant Management , MgSo4 – Magnesium sulphate.